ALCOHOLISM IN ARUBA

Oswald R. Wever

ALCOHOLISM IN ARUBA

KATHOLIEKE UNIVERSITEIT NIJMEGEN

ALCOHOLISM IN ARUBA

PROEFSCHRIFT

ter verkrijging van de graad van doctor in de geneeskunde
aan de Katholieke Universiteit te Nijmegen,
op gezag van de rector magnificus Prof. Dr. A. J. H. Vendrik,
volgens besluit van het college van decanen
in het openbaar te verdedigen
op vrijdag 11 november 1977 des namiddags te 4 uur

door

OSWALD RAYMOND WEVER

geboren op Aruba

1977
DRUKKERIJ VAN DENDEREN B.V.
GRONINGEN

PROMOTOR:

PROF. DR. F. J. A. HUYGEN

The publication of this thesis was made possible by grants of:
— the insular government of Aruba
— Stichting Bestrijding Alcoholmisbruik Aruba
— Lago Oil and Transport Company
— Stichting Alkohol Fonds
— various service clubs in Aruba
— STICUSA
— Stichting Hubrecht Janssenfonds & Schüffnerfonds

To my brothers and sisters in Alcoholics Anonymous in gratitude for what I received: the wish to serve and the ambition for humbleness

Pa mi esposa y yíunan

Pa mi mayornan

VOORWOORD

Dit proefschrift werd bewerkt op het Nijmeegs Universitair Huisartsen Instituut (Hoofd: Prof. Dr. F. J. A. Huygen), met medewerking van Drs. H. J. M. van den Hoogen, statisticus, en Drs. A. J. A. Smits, psycholoog. De computerbewerking vond plaats op het Rekencentrum van de Nijmeegse Universiteit.

Het veldonderzoek betreft een enquêtering van een aselecte steekproef uit de Arubaanse bevolking alsmede van een aantal behandelde alcoholisten en andere pathologische drinkers verricht in 1972-1973, en tevens gegevens uit statussen van alle tussen october 1969 en september 1972 in het San Pedro Hospital te Oranjestad behandelde pathologische drinkers.

Als enquêteurs en enquêtrices traden op:
A. Arends, S. Ashby, C. v. d. Berg-de Freitas, F. Celaire, J. Coronel-Peterson, C. Croeze-López, J. de Cuba, L. Ecury, J. Gumbs, L. Kelly, P. Kelly, C. Krottnauer, Zr. Ludovic, A. Martijn, A. Oduber, E. Oduber, R. Palm, O. Paris-Wever, Y. Spellen en M. de Windt.

Correctie en type-werk werden verzorgd door Mevr. F. Hoekstra en Mevr. A. M. K. E. Wever-Merks.

De figuren werden vervaardigd door de heren J. Brouwer en D. W. van der Beek van de grafische afdeling van de Interne Kliniek van het Academisch Ziekenhuis te Groningen, en de heer F. Hoffs verzorgde de fotografie.

CONTENTS

Chapter 1

INTRODUCTION

1.1. HISTORY

Alcoholism was already in 1836 pointed out as a major medical problem in Aruba by a Dutch Protestant clergyman visiting the island, the Rev. G. B. Bosch, who stated: "If one could only see the number of hogsheads of rum imported here every year from Curaçao, and consumed by this small population, excluding the notables of course, one would be absolutely amazed and inclined to believe that in proportion to the small population more strong drink is consumed here than at any other place in the world." (Bosch, 1836).

Nowadays rum is still one of the principal sources of beverage alcohol in Aruba, and still the impression exists that alcohol consumption levels are higher among lower than among higher social classes. Notwithstanding the long history and the urgency of the problem, there has been but a small number of publications on the subject. The oldest known publications, apart from Bosch, date back to 1955 (Henríquez, 1955), 1956 (Berkley, 1956), and 1957 (Braat, 1957; Turfboer, 1957). More recently there have been some publications in 1971 (Bijl, 1971; Janssen, 1971; Pijnenburg, 1971; Turfboer, 1971; Laclé, 1971; Vismans, 1971; De Windt, 1971; Oldenboom, 1971; Dalhuysen, 1971; Croes, 1971; Van der Voort, 1971; Driessen, 1971; Wever, 1971), 1975 (Wever, 1975), and 1976 (Wever, 1976, 1976a).

In September 1970 the First Aruban and Antillean Congress on Alcoholism was held in Aruba, on the occasion of the 15th anniversary of the local chapter of Alcoholics Anonymous; the bulk of publications on alcoholism in Aruba stems from this event.

1.2. PURPOSES OF THIS STUDY

This study is an epidemiological evaluation of alcoholism in Aruba. The purposes of this study are fourfold:

a) To determine prevalence rates of alcoholism, (gamma)-prealcoholism, problem drinking, social drinking and abstinence; this implies a *descriptive epidemiological approach*;

b) To determine prevalences of a number of mainly sociomedical, sociological and psychological characteristics correlated with the six diagnostic categories mentioned sub a); this also implies a *descriptive epidemiological approach*;

c) To test a number of mainly sociomedical, sociological and psychological theories - commonly considered as explanatory - as to their separate and combined capacity in explaining the occurrence of alcoholism and its precursor stages in Aruba; this implies an *analytical epidemiological approach*;

d) To evaluate treatment results in a number of therapeutic settings in a series of patients with alcoholism and its precursor stages treated from October 1969 through September 1972, and to compare these results with those in different therapeutic regimens in alcoholics treated from 1966 to October 1969 and with those mentioned in other studies; as well as to evaluate which of the factors found through analytical epidemiological research would be suitable for preventive and therapeutic measures; this implies *epidemiological operational research.*

From a *theoretical* point of view, it is important to evaluate whether alcoholism can be viewed as a single sharply defined and limited disease or whether there are detectable prealcoholismic syndromes in a random population sample, or whether alcoholism can be viewed as multidimensional, i.e. as a collection of diseases; moreover, it is important to investigate which of a number of theories can be denoted as significant in the shift from abstinence through social drinking to pathological drinking. From a *practical medical* point of view, it is important to determine the magnitude of alcohol problems in a community, in order to obtain basic information on which adequate planning of medical and social measures and projects can be executed for the near future - measures and projects aimed at the possible correction and prevention of these problems. More specifically, it is important to obtain reliable data concerning prevalences of chronic alcoholics, alcoholics, prealcoholics, gamma-prealcoholics and problem drinkers in a community, in order to set up or adjust preventive and therapeutic provisions for these specific groups of pathological drinkers. It is equally important, to obtain reliable data concerning those specific characteristics active in these

pathological drinkers, which possibly would react favorably to therapeutic or preventive measures, as well as concerning those which would not. It is evident too, that an evaluation of therapeutic results over a three years' period, also will have some significance for the planning of treatment programs in the future.

1.3. DESCRIPTION OF THE ISLAND AND ITS POPULATION

Aruba is a island in the Caribbean situated at 12°30′ north of the equator and 70° west of Greenwich, at 17 miles north of the Venezuelan peninsula of Paraguaná and 75 miles west of Curaçao. Geographically the island belongs to the South American mainland. This is illustrated by the fact, that the depth of the sea between Aruba and Paraguaná nowhere exceeds 300 feet, whereas depths of 4500 feet are found north of Aruba and between Aruba and Curaçao; moreover, in Aruba and Paraguaná the vegetation is practically identical. The highest point on the mainly level island is Yamanota hill, 570 feet high. In addition to the capital Oranjestad (or Playa, as it is called by Arubans) and the oil-town San Nicolás, there are four smaller population centers: Nort, Paradera & Tanqui Lender, Santa Cruz, and Sabaneta & Brazil. These six communities, together with their surroundings, divide the island's total area into six districts, called after these communities. By January 1, 1972, Aruba's population was 60.910. The distribution of the population over the six communities (districts) was at the time:

Oranjestad	16,711	(27.5%)
Nort	6,652	(10.9%)
Paradera & Tanqui Lender	4,695	(7.7%)
Santa Cruz	8,715	(14.3%)
Sabaneta & Brazil	8,126	(13.4%)
San Nicolás	15,901	(26.2%)

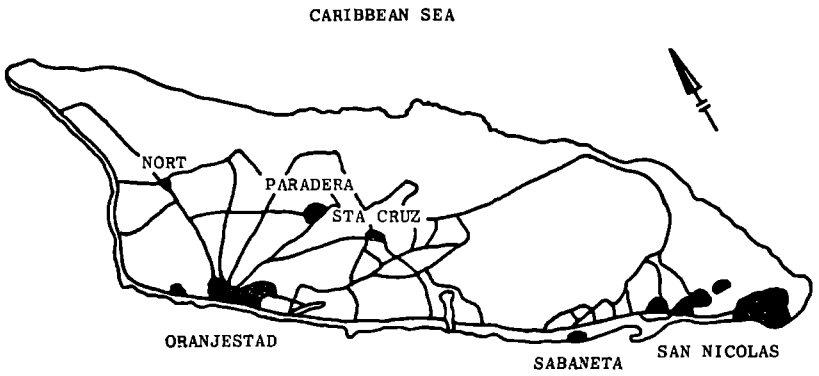
Figure 1.1. is a map of Aruba with a location of the principal population centers.

The island has an arid tropical climate despite a relatively high average air humidity of 73.9%; there is a low average annual rainfall of about 495 mm. The average air temperature is 27.5°C. with a low of 26°C. in January and a high of 29°C. in September. There

is a constant North-East tradewind the year around (Steenmeyer, 1957).

figure 1.1.

MAP OF ARUBA



Aruba is the third largest of the six islands of the Netherlands Antilles; the other five islands are Bonaire, Curaçao, St. Martin, St. Eustatius and Saba. Since 1954 these islands constitute a semi-autonomic part of the Kingdom of the Netherlands. The total population of the island has increased from about 51,000 in 1950 to 61,293 by December 1972, the year in which this study was carried out. The total area of the island is 190 km^2. Population density in 1972 was 323/km^2.

In 1972 the population consisted of about 80% Arubans and about 20% non-Arubans. The remaining 20%, however, includes people from about 50 countries or islands. This indicates the mixed, metropolitan composition of the total population. This 20% is mainly composed of Negroid people from the five other Dutch Antillean islands and other Caribbean islands and countries such as Surinam, Guyana, Venezuela, Colombia, Santo Domingo and Trinidad; there is also a considerable number of Dutch and U.S. citizens. In the nineteen-fourties however, the composition of the

population was different: about 50% Arubans and 50% other nationalities. The autochthonous Arubans can be considered as a trihybridic population with predominantly Amerindian and Caucasoid and to a lesser extent Negroid features. Arubans differ significantly in their ancestry from the inhabitants of other Caribbean islands, since they have a remarkable preponderance of Amerindian genes in their genetic pool.

The oldest known inhabitants of Aruba were Caiquetio Indians, a tribe originating from the Venezuelan mainland. After the arrival of the Spaniards around 1500 a great number of these Indians were carried off as slaves, mainly to Santo Domingo. After the Dutch took possession of the island in 1636, more and more Indians gradually established themselves in Aruba, not only from the Paraguaná peninsula and adjacent territories around the town of Coro in the present Venezuelan state of Falcón, but also from the Goajira region around the town of Maracaibo in the present Venezuelan state of El Zulia and adjacent regions in Colombia. The Indian traits in comtemporary Arubans can in all probability be attributed mainly to these later Indian settlers (Hartog, 1953). The island was closed territory to whites for many decades. After 1754 more and more people of different European and Latin American nationalities settled in Aruba and intermingling of races has continued ever since. A comparatively large number of Indians was smuggled into Aruba as "red slaves" and more than 500 "true" Indians were still to be found in the population at the beginning of the 19th century. The present Aruban population has developed from and alongside the Amerindians (Hartog, 1953). Negro slaves were also present in Aruba in the past three centuries, although their numbers were never large, due to the fact that there were but few plantations on the island. Their influence on the development of the contemporary Aruban population has been negligible, since the Indian and Caucasoid elements have always remained predominant; in Curaçao, for example, the proportion of Negroes in the population, was approximately ten times greater (Hartog, 1953).

The language spoken by Arubans is Papiamento, a typical composite language mainly based on creolisation of 16th and 17th century Spanish and Portuguese by native Indians and Negro slaves, with

the main impact of creolisation in its grammar, syntaxis and intonation, and a predominance of Spanish and Portuguese in its vocabulary; beside Spanish and Portuguese influences there is a considerable proportion of words of Dutch, English, Amerindian and African origin in Papiamento vocabulary.

As previously mentioned, alcoholism was already pointed out as a major problem in Aruba in 1836 (Bosch, 1836). Since alcoholism only constituted a minor problem on the five other Dutch Antillean islands, its greater prevalence in Aruba for many decades has been ascribed to "the Indian blood of Arubans", and this statement has been officially repeated as late as 1971 by the former director of the Public Health Department (Bijl, 1971).

It is a common observation, that the prevalence of alcoholism is high among Amerindians, though it has never been proven that this is due to genetic factors. Against this hypothesis can be argued, that among Chinese - another Mongolian sub-race like Amerindians - in the USA the prevalence of alcoholism is low (Chu, 1972).

1.4. DEFINITIONS

1.4.1. Alcohol

Ethyl alcohol (synonyms: ethanol, alcohol) is probably the most widely used psychoactive drug in the world, with the exception of caffein. The term "drug" implies any substance, other than those required for the maintenance of normal health (as opposed to the correction of a disease), which by its chemical nature alters the structure or function of a living organism (Kalant & Kalant, 1971). The term "psychoactive drug" implies any drug which is used primarily for its effect on mood, perception and consciousness, regardless of what the normal medical use of such substances may be (Kalant & Kalant, 1971).

Alcohol is formed by the deliberate or accidental fermentation of fruits, grains, potatoes or other foods rich in sugar or starch. Depending on the methods of fermentation, the nature of the starting material, and the subsequent application of distillation, alcohol is found in a large variety of beverages ranging in strength from weak beers through wines of varying potency to the distilled liquors. Alcohol (ethanol) has as its chemical formula: C_2H_5OH.

Though there are minor differences in effects of alcoholic beverages resulting from the presence of ingredients other than ethanol itself, almost all authorities agree that by far the most important share of the effects of all alcoholic beverages is explicable entirely in terms of alcohol content.

Together with opiates (e.g. opium, morphine, heroin, codeine, pethidine), volatile solvents (e.g. isoamyl acetate, ethyl acetate), chloral hydrate and paraldehyde (both are alcohol derivatives), barbiturates, major tranquillizers (e.g. reserpine, phenothiazines), and minor tranquillizers (e.g. meprobamate, chlordiazepoxide), alcohol belongs to the category of depressant or sedative drugs.

1.4.2. Categories of psychoactive drugs

Psychoactive drugs can be classified (Kalant & Kalant, 1971), into three major groups:

a) *Depressant or sedative drugs.* These are drugs, which decrease the state of alertness and by this means diminish the impact of the outer environment upon the thoughts and feelings of the user.
b) *Stimulant drugs.* These are drugs, which increase the state of arousal, so that the brain is exposed to much larger input of information from the environment, and mental processes are speeded up. Drugs of this type include: amphetamines, cocaine, caffeine, strychnine and methylphenidate (Ritalin).
c) *Distorters of consciousness and perception (psychedelics).* This category includes several different types of drugs which have varying degrees of depressant or stimulant action but all act primarily to alter the *quality* rather than the *intensity* of the user's perception of events occurring within himself and in his outer environment. Drugs of this type include: LSD, mescaline, psilocybin, myristicin, elemicin, atropine and its derivatives, muscarine, cannabis preparations such as marihuana and hashish and the synthetic tetrahydrocannabinol (THC).

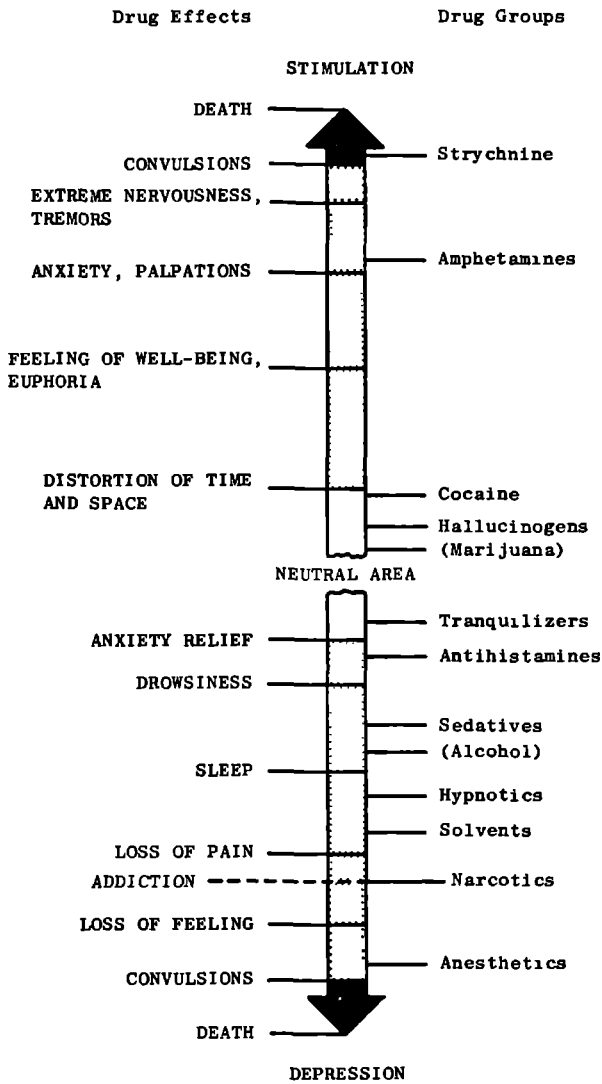
1.4.3. Continuum of drug actions and effects

Jones, Shainberg and Byer (1970) only distinguish depressant and stimulant drugs, and include psychedelics in the category of stimulants. They describe a continuum of drug actions and effects as suggested by Dr. Robert W. Earle of the University of California; this is visualized in *fig. 1.2.*

As shown in *fig. 1.2.* the continuum of drug effect reaches over-stimulation and death at one extreme, and severe depression and

figure 1 2

CONTINUUM OF DRUG EFFECTS AND ACTIONS



death at the other. The drug groups are placed in *fig. 1.2.* according to the kind and degree of effects they produce when a normal therapeutic dose is consumed by an individual. If dosages are increased, any of the drugs in *fig. 1.2.* may produce the complete range of effects of stimulation (in the case of stimulant drugs) or of depression (in the case of depressant drugs).

The more commonly used drugs, such as marihuana or the barbiturates, are close to the center of the chart, when used in small dosages. The strong preference for these drugs lies in the ability of an individual to control the amount and consequently the relative effect of the drug. However, this ability to control drugs is offset in the case of the *depressant* drugs, when *addiction* levels are reached, cf. *fig. 1.2.;* then, regardless of the user's desire or emotional state, a small but increasing dose is regularly required to keep the body from entering into a withdrawal syndrome. Jones, Shainberg and Byer (1970) state that stimulant drugs do not seem to have the addictive properties of the depressants. This view is contradicted by Kalant & Kalant (1971), who state that in both stimulant drugs and depressant drugs a withdrawal syndrome, and hence addiction, is possible. They describe the withdrawal reaction as a probable result of overcompensation of the central nervous system to the effects of a drug; in the case of a depressant drug this overcompensation implies overstimulation by the central nervous system (CNS), and in the case of a stimulant drug this overcompensation implies extreme depression by the CNS. Nevertheless, the most serious withdrawal reactions are those caused by removal of depressants such as alcohol, opiates and barbiturates. The full-blown picture of delirium tremens in alcoholism is almost identical to the withdrawal syndrome in barbiturate addiction. Withdrawal syndromes in excessive users of stimulant drugs are generally less impressive, since they consist mainly of excessive sleeping, though in a few instances e.g. amphetamine withdrawal has been followed by suicide during the severe depression which may occur.

1.4.4. Alcoholism

For the purpose of this study, alcoholism is defined as the condition in which an individual repetitively consumes beverage alcohol

to such an excess that he gradually develops an increasing tolerance to ethanol and regularly experiences withdrawal symptoms when reducing or abruptly stopping his alcohol intake; these withdrawal symptoms may or may not be accompanied by other physiological and psychological symptoms preceding, concurrent with, or following alcohol intake; other social and psychological characteristics of problem drinking may also be present in the conjoint symptomatology of alcoholism.

Tolerance to ethanol is the condition in which an individual in the course of time gradually needs larger quantities of beverage alcohol to obtain the same level of sedative, desinhibitive, euphoric and other effects of ethanol.

Withdrawal symptoms include: general discomfort, irritability, restlessness, tremors, sweating, nausea, vomiting, palpitations, anxiety, insomnia and other sleep disturbances, hyperexcitability, depression, tenseness, convulsions, auditive and visual hallucinations, and delirium tremens. Characteristically, these withdrawal symptoms tend to fade away or disappear when alcohol intake is resumed or increased; this in contrast with the symptoms of intoxication which tend to increase when alcohol intake is resumed.

Symptoms of alcohol-intoxication include: alcohol fragrance of breath, wide pupils, swollen reddened face, light conjunctival injection, dysarthric speech, sweating, vomiting, light motor disturbance of fine movements to heavy cerebellar atactic gait, emotional lability, pseudovirile obtrusiveness, endless sometimes incoherent thought-associations, megalomania, euphoria, depression, aggressiveness, reduction of consciousness to profound coma, convulsions, and hypoglycemia.

The *other physiological and psychological symptoms* include: loss-of-control, early morning drinks to cope with a hangover or withdrawal, blackout, frequency of intoxication of once a week or more, alcoholic gastritis, alcoholic polyneuritis, alcoholic psychosis, craving, and drinking to cope with intrapersonal or interpersonal problems (psychological dependence).

This definition is compatible with the diagnostic criteria as proposed by the Criteria Commitee, National Council on Alcoholism (NCA) (1972) as well as with the symptomatology described by

Jellinek (1960), and with the definition of alcohol addiction as classified under section 303.2 in the "Diagnostic and Statistical Manual of Mental Disorders" of the American Psychiatric Association (1968). A concise description of Jellinek's symptomatology of alcohol addiction is given by Jones et al. (1970) and by Van Epen (1974).

The main differences between Jellinek's symptomatologic definition and the one employed in this study, are:

a) The loss-of-control phenomenon is *not* considered as the paramount pathognomonic symptom of alcohol addiction, and this is in agreement with the diagnostic level *2* ("probable, frequent, indicative") assigned to this symptom by the Criteria Committee (1972);
b) The withdrawal syndrome *is* considered as the central, pathognomonic criterion, and this is in agreement with the diagnostic level *1* ("classical, definite, obligatory") assigned to it by the Criteria Committee (1972).

This difference in definition is maintained, notwithstanding Keller's statement (1972), that loss-of-control still is to be considered as pathognomonic, for the following reasons:

a) to keep this study's definition in concordance with the NCA criteria (Criteria Committee, 1972);
b) because in studies of other types of drug-addiction the withdrawal syndromes are also considered as obligatory criteria;
c) so far there is no complete agreement as to whether the loss-of-control phenomenon is a *physical* symptom pertaining to the withdrawal syndrome, or a conversion phenomenon, a sign of mental compulsiveness, i.e. a *psychological* symptom (Jellinek, 1952; Plaut, 1967; Criteria Committee, 1972).

The possible triggering of a presumed biochemical abnormality basic to addiction by one of the first drinks on a drinking occasion, as an explanation for loss-of-control, has never been definitely (dis)proven (Keller, 1972). Still, loss-of-control is generally considered as the pathognomonic sign of alcoholism, the "point of no return", by Alcoholics Anonymous (AA) and by a considerable number of experts in alcohol studies; as a matter of fact, this symptom was primarily derived from field observations by AA (Keller, 1972).

Keller (1972) states that without loss-of-control there is only a prealcoholismic phase but not alcoholism. According to the definition employed in this study, the statement is: without firm withdrawal syndrome and in the presence of loss-of-control, there is only a prealcoholismic phase, not alcoholism.

Another definition of alcoholism has been given by Schmidt & De Lint (1970): Alcoholism is the condition in which an individual consumes alcohol in excess of a daily average of 15 cl. (= 150ml) of absolute alcohol. This definition was derived from the clinical observation in several countries, that alcoholics generally drink in excess of 15 cl. of absolute alcohol daily (Péquignot, 1958; Lelbach, 1966; Schmidt & Popham, 1968; Wilkinson et al. 1969). It is a definition of a strictly quantitative nature, based on empirical findings.

1.4.5. Problem drinking

For the purpose of this study, problem drinking is defined as the condition in which an individual repetitively consumes beverage alcohol to such an excess, that physical, psychological or social harm is caused to that individual or to significant others; tolerance and the withdrawal syndrome may or may not be present. According to Plaut (1967), from whose work this definition was derived, this is a rather broad definition which *includes* the condition called "alcoholism". It is easily understood, that according to this definition all alcoholics can be considered problem drinkers since by their excessive drinking they do cause physical, psychological or social harm to themselves or to significant others. On the other hand, only those problem drinkers are to be considered as alcoholics, who have developed increasing tolerance to ethanol and experience withdrawal symptoms.

According to Keller (1972) problem drinkers showing loss-of-control should be considered as alcoholics; according to the definition employed in this study these would have to be considered as prealcoholics.

In between *problem drinking* and *alcoholism* however, there can be found individuals who do not convincingly nor completely fulfill the criteria for the diagnosis of alcoholism, but only to a certain extent. These individuals can be considered as "alcoholics *in statu*

nascendi". According to the Criteria Committee of the NCA (1972) for these individuals the diagnosis should be: *suspected alcoholism.*

In this diagnostic category two types can be distinguished:

a) loss-of-control-prealcoholism or gamma-prealcoholism,
b) (withdrawal syndrome-)prealcoholism.

1.4.6. Gamma-prealcoholism

For the purpose of this study, gamma-prealcoholism is defined as the condition in which an individual shows *loss-of-control* without withdrawal symptoms. Some of the other previously mentioned physiological and psychological symptoms pertaining to the diagnosis of alcoholism may also be present. The name for this definition as used in this study, has been devised partially as a tribute to Jellinek's typology of alcoholism (Jellinek, 1960).

1.4.7. Prealcoholism

For the purpose of this study, prealcoholism is defined as the condition in which an individual only to a certain extent shows some indication of withdrawal symptoms, and at the same time absence of loss-of-control and of the other previously mentioned physiological and psychological symptoms of alcoholism.

1.4.8. Social drinking

For the purpose of this study, normal social drinking is defined as the condition in which an individual regularly or irregularly consumes beverage alcohol without ever showing any of the symptoms pertaining to alcoholism, gamma-prealcoholism, prealcoholism or problem drinking.

1.4.9. Abstinence

For the purpose of this study, abstinence is defined as the condition in which an individual never consumes beverage alcohol.

1.4.10. Alcoholism-with-complications

For the purpose of this study, alcoholism-with-complications is defined as the condition in which an individual responds to the diagnosis of alcoholism, and at the same time shows one or more of the known medical and psychiatric alcoholismic complications. To

these complications pertain: alcoholic fatty degeneration of the liver; alcoholic hepatitis; alcoholic liver cirrhosis (Laennec's cirrhosis); alcoholic pancreatitis; acute and chronic alcoholic gastritis; Mallory-Weiss syndrome; alcoholic myocardiopathy; Zieve syndrome; alcoholic hypoglycemia; beriberi; pellagra; scorbutus; anemia (hypochromic, normocytic, macrocytic, hemolytic with stomatocytosis, folic acid deficient); thrombocytopenia; leukopenia; alcoholic myopathy; alcoholic adrenocortical hypofunction; alcoholic polyneuritis; Korsakoff syndrome; Wernicke syndrome; toxic amblyopia; blackout (palimpsest); alcoholic convulsions; alcoholic hallucinosis; delirium tremens; retrobulbar neuritis; pachymeningitis haemorrhagica interna; alcoholic pseudotabes; alcoholic pseudoparalysis; alcoholic cerebellar degeneration; alcoholic coma; Marchiafava-Bignami's disease (post-mortem diagnosis); central pontine myelinolysis (post-mortem diagnosis); alcoholic cerebral degeneration; alcoholic dementia; alcoholic paranoia; alcoholic depression; suicidal behavior; alcoholic aggressiveness. (This enumeration does not pretend to be complete).

According to Jellinek's study (1951), the proportion of alcoholics-with-complications among all alcoholics in the U.S.A. would be about 25%. In a later study Jellinek (1959) revised this proportion to 18.75%. Other data from ten other countries however (Keller & Efron, 1955), make mention of varying figures for this proportion, varying from 25% in England, Finland, Norway, Denmark, Sweden and Canada, through 50% in France and Australia, 67% in Switzerland, to 100% in Chile.

1.4.11. Chronic alcoholism

For the purpose of this study, and in agreement with data from the literature, chronic alcoholism is defined as the condition in which an individual fulfills the criteria for the diagnosis of alcoholism, and moreover shows the following symptoms: drinking bouts of extremely long duration, e.g. weeks; *decreasing* tolerance to alcohol; frequent morning drinks; marked ethical deterioration; impairment of thinking; regression to the lowest social levels (Skid Row); consumption of "technical products" such as bay-rum, rubbing alcohol, methanol and other types of denaturated alcohol; psychomotor inhibition; and obsessive drinking (Jellinek, 1952, 1960, 1962). According

to various authors and studies, this category only comprises 3% to maximally 10% of all alcoholics (Ford, 1956; Fox, 1961; Block, 1962; National Council on Alcoholism, 1970; Albrecht, 1973).

1.4.12. Jellinek's symptomatology

Jellinek has described a number of symptoms frequently found in the drinking histories of alcoholics (Jellinek, 1960).

A review of the most important symptoms is given as follows:

a) *Increase of tolerance.* This was described above.
b) *Withdrawal syndrome.* The withdrawal symptoms were also described above.
c) *Blackout:* partial amnesia for past events during a drinking bout.
d) *Loss-of-control:* the individual cannot stop drinking alcohol after the first or one of the first drinks at the start of a new drinking bout, but is innerly driven to continue drinking alcohol compulsively, until he is intoxicated or until he experiences untoward effects of drinking such as for instance vomiting.
e) *Drinking to cope with problems; escape drinking:* the individual drinks to cope with or to escape anxiety, tenseness, restlessness, fatigue, sorrow, depression, stress, or specific intrapersonal or interpersonal problems.
f) *Drinking in drinking bouts:* the individual's drinking pattern is characterized by periods of heavy drinking alternated by periods abstinence; in the course of time drinking bouts progressively tend to grow longer and periods of abstinence progressively shorter.
g) *Craving:* the individual frequently experiences a nearly unsupportable yearning for beverage alcohol, even when he has been sober for a considerable period.
h) *Early morning drink:* the individual frequently consumes beverage alcohol to cope with a hangover or with light or severe withdrawal symptoms.
i) *Hiding supplies of beverage alcohol.*
j) *Pretending to dislike beverage alcohol.*
k) *Clandestine drinking.*

Of these symptoms the ones sub b), c), d), e), g), and h) were included in the questionnaires used in this study. Tolerance (a) was excluded, since there is increase of tolerance at the beginning of alco-

holism, followed by a steady level of high tolerance, and ultimately followed by a decrease of tolerance in chronic alcoholism, according to Jellinek (Jellinek, 1952, 1960, 1962), and because tolerance not only is a sign of drug addiction but also of drug habituation (Seevers, 1962; Walsh, 1973). *Drinking in drinking bouts* as diagnostic criterion is included in a question in the employed questionnaire concerning the annual frequency of intoxication; as a descriptive criterion it was only tested in questionnaire B (cf. Appendix A) to distinguish between the various types of pathological drinking.

1.4.13. Jellinek's phaseology

Jellinek's phaseology expresses Jellinek's (Jellinek, 1952, 1960, 1962) opinion, that the alcoholismic symptoms tend to appear more or less in a certain order of succession. These *phases* are:

a) the *prealcoholismic* phase. Drinking gives marked relief (psychological dependence); increase of tolerance;
b) the *prodromal* phase. Frequent blackouts; clandestine drinking; preoccupation with alcohol; drinking before going to parties; gulping drinks; avoiding conversation about drinking; guilt feelings regarding drinking;
c) the *critical* phase. Loss-of-control; rationalization of one's alcohol consumption; hiding supplies of beverage alcohol; periods of total abstinence; changing drinking habits in order to obtain control over drinking again;
d) the *chronic* phase. Drinking bouts of long duration; early morning drinks; tremors; decrease of tolerance.

Van Epen (1974) states that it is doubtful whether these phases really exist. It was not possible to find good agreement between Jellinek's phaseology and the definitions of alcoholism, gamma-prealcoholism, prealcoholism, and problem drinking, employed in this study.

1.4.14. Jellinek's typology

Jellinek's typology (1960) expresses Jellinek's view that there are five distinguishable *types* of alcoholism:

a) *Alpha-alcoholism.* Psychological dependence on the effects of alcohol; absence of physical dependence and hence of the withdrawal syndrome;

b) *Beta-alcoholism.* Presence of somatic diseases caused by excessive consumption of beverage alcohol, such as for instance dietary deficiencies, gastritis, alcoholic steatosis or cirrhosis of the liver; absence of physical dependence;
c) *Gamma-alcoholism.* Loss-of-control; increasing tolerance; physical dependence and hence withdrawal syndrome; blackout; this is considered the "classical" syndrome of alcohol addiction;
d) *Delta-alcoholism.* Daily consumption of very large quantities of beverage alcohol; absence of loss-of-control; absence of physical dependence; stopping alcohol consumption is impossible (inability to abstain);
e) *Epsilon-alcoholism.* Periodic drinking patterns, such as for instance dipsomania.

It was not possible, to find good agreement between Jellinek's typology and the definitions of alcoholism, gamma-prealcoholism, prealcoholism and problem drinking, employed in this study. Still it may be stated, that there exists fair agreement between this study's definition of problem drinking on the one hand, and Jellinek's alpha-alcoholism, beta-alcoholism, and epsilon-alcoholism on the other hand; there is also some agreement between this study's definition of alcoholism (alcohol addiction) and Jellinek's gamma-alcoholism and delta-alcoholism; there is lesser agreement between this study's definitions of gamma-prealcoholism and prealcoholism and Jellinek's gamma-alcoholism.

1.4.15. Addiction

Seevers (1962) has given enlightening definitions of drug addiction and drug habituation.

Drug *addiction* is a state of periodic or chronic intoxication produced by the repeated consumption of a natural drug (such as for instance alcohol) or of a synthetic drug (Seevers, 1962).

Its characteristics are:

1) an overpowering desire or need (compulsion) to continue taking the drug and to obtain it by any means;
2) a definite tendency to increase the dose (i.e. increase of tolerance to the drug);
3) a psychological dependence on the effects of the drug;

4) a definite physical dependence on the effects of the drug and hence a withdrawal syndrome;
5) detrimental effects on the drug user *and* on society.

1.4.16. Habituation

Drug *habituation* is a condition resulting from the repeated consumption of a drug (Seevers, 1962). Its characteristics are:

1) a desire (but *not* a compulsion) to continue taking the drug for the sense of improved well-being or the effect that it produces;
2) little or no tendency to increase the dose;
3) some degree of psychological dependence on the effects of the drug;
4) absence of physical dependence and hence of a withdrawal syndrome;
5) detrimental effects, if any, primarily on the drug user, and less on society.

It is evident, that the main difference between addiction and habituation is to be found in the presence or absence of physical dependence, and hence of the withdrawal syndrome.

It is evident too, that the definition of alcohol addiction as employed in this study, is in agreement with Seevers's (1962) definition of addiction.

It is clear too, that this study's narrower definition of problem drinking is in agreement with Seevers's (1962) definition of habituation, since it considers withdrawal syndromes as evidence of addiction and not of habituation.

This study's definitions of gamma-prealcoholism and prealcoholism more or less form transitional stages between habituation and addiction, considering the compulsive characteristic of the loss-of-control phenomenon in gamma-prealcoholism, and the unconvincing presence of withdrawal symptoms in prealcoholism.

The four mentioned conditions - problem drinking, prealcoholism, gamma-prealcoholism, and alcohol addiction (or *alcoholism*) - can therefore be considered as consecutive steps or phases in what adequately has been called "the alcoholismic learning process" by Keller (1972), in the sense that in the course of time both body and mind of the drinker learn to present the symptoms pertaining to these phases.

1.4.17. Tolerance and physical dependence

Tolerance can be due (Kalant & Kalant, 1971; Walsh, 1973) to at least three different mechanisms: metabolic tolerance, cellular tolerance and central nervous system (CNS) tolerance.

a) *Metabolic tolerance.* This consists of an increase in the activity of the mechanisms in the liver and other tissues by which a drug such as alcohol is metabolically destroyed. From the point of view of the user of the drug c.q. alcohol, it is simply equivalent to taking a smaller dose of the drug, because if more of the drug is catabolized in a given time, then less of it remains in the body to exert its characteristic action. Chronic administration of ethanol can induce increases in the rate of alcohol metabolism in alcoholic and non-alcoholic subjects (Lieber, 1973, 1973a; Walsh, 1973).

However, the claim that prolonged alcohol ingestion could induce an increase in hepatic alcohol dehydrogenase activity (as the primary enzyme responsible for ethanol oxidation) has not been consistently reproduceable (Walsh, 1973).

Neither has this been the case with alcohol dehydrogenase isoenzyme in human liver about which Von Wartburg et al. postulated that it would be related to an increased rate of alcohol metabolism (Von Wartburg et al. 1965, cited by Walsh, 1973).

The microsomal ethanol oxidizing system (M.E.O.S.) appeared to be inducible after prolonged alcohol ingestion in rats (Lieber, 1973, 1973a; Walsh, 1973).

Chronic alcohol ingestion in rats induced a proliferation of the smooth endoplasmic reticulum, and in rats as well as in man it induced hyperactivity of certain microsomal drug metabolizing enzymes. This inducibility has been suggested as a possible basis for alcoholic tolerance and the cross-tolerance in alcoholics to certain sedative and hypnotic drugs (Lieber, 1973, 1973a; Walsh, 1973). Evidence has accumulated that oxidation by the microsomal fraction may be due to a peroxide generating system coupled with catalase arising as an artifact of tissue disruption (Walsh, 1973).

b) *Cellular tolerance.* This implies: increased release of neurotransmitter substances such as catecholamines (Mendelson, 1970, 1970a; Walsh, 1973) by chronic alcohol ingestion; induction of

liver aldehyde dehydrogenase by chronic alcohol ingestion, with consequent lowering of tissue acetaldehyde; inhibition of acetylcholine release in rat brain by ethanol; stimulation or inhibition of Na-K-adenosine triphosphatase mediated transport of Na^+ and K^+ ions by ethanol in guinea pig and rat brain and other tissues, as well as of this enzyme's activity in erythrocytes of alcoholics (Walsh, 1973).

c) *CNS tolerance.* This implies an adaptive change in the CNS which compensates for the effects of the drug and thus renders the user less sensitive to it. For example, chronic use of depressants, leads to a compensatory increase in the excitability of neurons, offsetting the decrease in excitability produced by the depressant drugs. The user must then take a larger dose in order to achieve the depressant effect he seeks. When the drug is removed, the increased excitability of the neurons remains for some time. But instead of being a beneficial compensation for the effects of the drug, it is actually a disturbing phenomenon because it leaves the neurons, from which the drug was withdrawn, excessively responsive to normal stimuli. As a result the individual becomes hyperexcitable, jittery, unable to sleep, and bothered by various signs of overactivity of the autonomic nervous system, including sweating, nausea, palpitations, hallucinations, tremors, and convulsions. Most chronic users of depressants learn to recognize the early symptoms of such withdrawal reactions; they learn that by taking more of the drug they can cause these symptoms to diminish or disappear. In this case they are using the drug as a form of treatment for the withdrawal syndrome which was caused originally by the drug, and in this state they have developed *physical dependence* on the drug, in the sense that when decreasing their drug intake they suffer the corresponding withdrawal syndrome (Mendelson, 1970, 1970a; Kalant & Kalant, 1971; Victor, 1973; Victor & Wolfe, 1973; Walsh, 1973). It is hypothesized, that withdrawal of alcohol (and other depressants) induces an increase in excitability of nervous pathways that is masked by the depressant action of alcohol on the same pathways, *or* that alcohol induces depression through activation of suppressive pathways (Mendelson, 1970a). Upon removal of alcohol (and other

depressants) there is a consequent "rebound hyperexcitability" of the depressed functional systems. This is due to the "denervation supersensitivity" or "disuse supersensitivity" which occurs in the CNS during chronic CNS-depression by depressant drugs (Mendelson, 1970a).

Victor et al. have found that two factors in particular, hypomagnesemia and respiratory alkalosis, are consistently associated with alcohol withdrawal symptoms (Victor, 1973; Victor & Wolfe, 1973).

This has also been stated by other authors (Mendelson et al., 1959). Convulsions combined with hypomagnesemia have been reported in infants (Friedman et al., 1967; Savage & Mc Adam, 1967; Paunier et al., 1968) and convulsions are a well known symptom of alcohol withdrawal.

Both central and peripheral nervous system irritability have been described in states of hypomagnesemia. In several animal species, hypomagnesemia has been associated with the occurrence of seizures. In man it has been associated with tremor, twitching, seizures, carpopedal spasm, and hallucinations, while some of these symptoms can be reversed by the administration of magnesium (Victor, 1973).

Respiratory alkalosis in alcohol withdrawal is probably caused by hyperventilation (Victor, 1973). Respiratory alkalosis may be accompanied by tremors, carpopedal spasm, disorientation, hallucinations, hyperreflexia, muscular irritability, hyperpyrexia, and seizures (Victor, 1973). Respiratory alkalosis and hypocapnia both may lead to increased nervous system irritability (Victor, 1973). Hypocapnia leads to cerebral vasoconstriction and hence to cerebral hypoxia, thereby potentiating the effect of alkalosis on neural excitability, and accounting for many alcohol withdrawal symptoms (Victor, 1973). Alkalosis may cause hypomagnesemia by possibly causing a shift of magnesium into bone and other intracellular sites.

Possibly, removal of the depressant effect of alcohol on the respiratory center (during withdrawal) is followed by a "rebound" phenomenon, resulting in increased sensitivity of this center to carbon dioxide and hyperventilation (Victor, 1973).

1.5. EPIDEMIOLOGY AND ITS CHARACTERISTICS

Epidemiology is the science which studies the characteristics and determinants of health, disease and death as collective phenomena (Van Zonneveld et al., 1972). Epidemiology can be subdivided as follows (Van Zonneveld et al., 1972):

1) *Descriptive epidemiology:* the study of the prevalences and incidences of disease, death or health and of characteristics associated with these three phenomena;
2) *Analytical epidemiology:* the study which has as its purpose to detect the causes of disease and death;
3) *Operational research:* the study which has as its purpose to determine the health *needs* in a community by the application of scientific methods, techniques and instruments on problems related to the functioning of a public health system, in order to present optimal solutions for these problems to those people in charge with the control of that public health system. There is a difference between health *needs* and health *demands.* Health *demands* are (subjective) demands proposed by lay people. Health *needs* are needs objectively assessed by experts.

Epidemiology is closely related to *ecology,* the science which studies the relationships between organisms and their *environment,* e.g. the interaction between man and his environment with regard to disease.

In this study two types of environment are studied:

a) The *microclimate:* the environment of the family unit and the extended family unit;
b) The *macroclimate:* the environment of the community as a whole, including the working situation.

Environment can also be subdivided into:

1) *Material environment:* the physical, chemical and biological environment;
2) *Immaterial environment:* the environment of human relations.

In this study only a few factors in the material environment are studied (possible genetic factors; alcohol as toxic agent; physical dependence; somatic complications of alcoholism) next to an extensive number of factors in the immaterial environment (Chapters 3, 4, 5, and 6).

Human ecology considers the development of a community as a process of adaptation of a population to its environment, taking into account the technical systems and organization patterns by which that adaptation is effectuated. The functioning of a community within its environment can be considered as a complex *ecologic model*, which is composed of four interdependent elements: technology, social structure, biological environment and population (Van Zonneveld, 1972). This ecologic model can be extended to an *ecologic model of public health*, as shown in *fig. 1.3.* (Van Zonneveld, 1972).

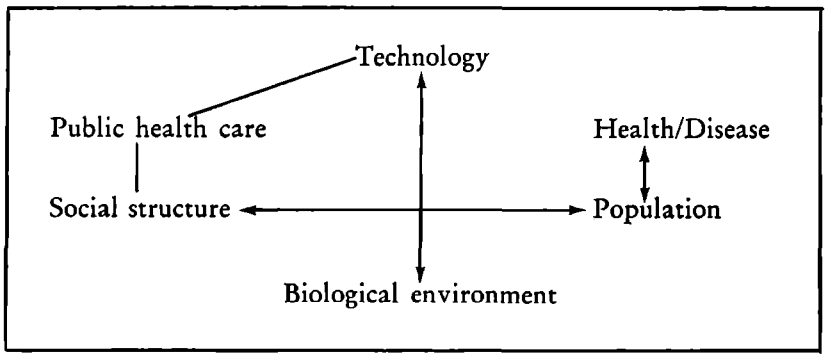


Fig. 1.3. Ecologic model of public health

Some remarks with regard to *fig. 1.3.*

— *Social structure* implies man's endeavors to realize material and immaterial improvement of the community.
— *Technology* implies the technical skills applied or applicable to such improvements.
— *Biological environment* comprises water, air and soil, inclusive fauna and flora, i.e. the primary necessaries of life such as food, water, light, housing, clothing and heating.
— *Population* implies the collection of individuals and their structure according to e.g. age, sex, social class.
— *Public health care* implies all measures and provisions aimed at the promotion of health and the prevention or cure of diseases. Public health care pertains both to technology (medical skills) and to social structure (endeavors to realize material and immaterial improvement of the community).

1.5.1. Descriptive epidemiology

Descriptive epidemiology tries to give a picture of disease or health in a population and its subdivisions. Answers are sought for such questions as: "which disease or phenomenon? in which individuals? where? when?" To answer the question "which disease?" implies the application of *diagnostic criteria* in order to determine the prevalence of these diagnostic criteria (f) in a population (N). $\frac{f}{N}$ is known as the *epidemiological fraction.*

Diagnostic criteria must meet the following requirements:

a) They must be defined operationally;
b) They have to be representative characteristics of the disease to be studied;
c) It must be possible to use them also to classify the degree of severity of a disease.

Tests with which diagnostic criteria are tested must meet the following requirements:

a) They must be as simple as possible, since they must be applicable for mass screening (*simplicity*);
b) They must deliver reproduceable results (*repeatability*);
c) They must have a high *specificity*, i.e. they must detect *only* those persons with a specific disease (Sturmans & Mulder, 1976, 1976b);
d) They must have a high *sensitivity*, i.e. they must detect the largest possible proportion of patients with one specific disease.

Repeatability of a test depends on:

1) *Intrinsic accuracy of a measurement*, e.g. errors inherent in measuring instruments;
2) *Constancy of the characteristic* measured, e.g. variations of characteristics within individual patients in time and under varying conditions;
3) *Researcher's ability to handle measuring methods and to note and interpret his observations;*
4) *Intra-observer variation*, i.e. varying perceptive faculty and judgment of an individual researcher in time;
5) *Inter-observer variation*, i.e. varying perceptive faculty and judgment between two or more researchers.

In the *population* to be studied in descriptive epidemiology the following characteristics are generally tested:

a) *Personal characteristics:*
   - — age
   - — sex
   - — social class
   - — income

b) *Space:*
   - — country
   - — region
   - — province
   - — urban/rural domicile
   - — housing conditions

c) *Time:*
   - — hours
   - — days
   - — weeks
   - — months
   - — years

Measures of disease, death and health can all be represented in the form of frequency distributions. The following measures can be applied:

1) *Rate:* $\frac{\text{number of patients (or deaths) in a certain period}}{\text{total population on a certain moment in time}}$

   Rate is usually expressed in percent or in $^0/_{00}$, per 10,000 or per 100,000 of total population (Sturmans & Mulder, 1976a).

2) *Prevalence:* $\frac{\text{number of patients (or deaths) on a certain moment in time}}{\text{total population}}$

3) *Point prevalence* is the *rate* at a certain moment in time (Oliemans, 1969).

4) *Period prevalence* is the *rate* in a certain period.

5) *Incidence* is the number of disease cases in a certain period *or* the number of persons getting a disease at least once in that period (Sturmans & Mulder, 1976a).

6) *Specific rate* is the *rate* for a specific population segment.

7) *Gross rate* is the *rate* for the total population; this can be viewed as the sum of the weighed specific rates, with as weighing coefficients the relative frequencies for the population segments, i.e.

each of the specific rates contributes to the gross rate proportionate to the share which the specific population segment has in the total population.

The following *types of epidemiological research* can be distinguished:

1) *Transversal* study: the distribution of disease or health and their determinants is studied at a certain moment or during a certain period in time; this represents a cross-section in time, and determines point prevalence and period prevalence.
2) *Longitudinal* study: repeated transversal investigation in the course of time.
3) *Cohort* study: repeated transversal investigation in the course of time, for certain birth cohorts (generally 5-year periods), by which procedure age-specific morbidity can be determined per birth cohort.

The present study represents a transversal study.

Another classification of types of epidemiological research is as follows.

a) Study of *health statistics:* existing data are used to study the distribution of disease or health and their determinants.
b) *Health examination survey:* physical examination, laboratory procedures, etc., used in a population to study the distribution of disease or health and their determinants.
c) *Health interview survey:* interview techniques (questionnaires, etc.), used in a population to study the distribution of disease or health and their determinants.

The present study mainly represents an interview study, combined with data from health statistics.

Epidemiological studies can be performed in:

1) The total population;
2) A random sample of the total population.

The present study deals with a random sample of Aruba's population in 1972.

1.5.2. Analytical epidemiology

Analytical epidemiology tries to give answers to the question: "which are the causes of (a certain) disease or death?" Knowledge of causes of disease/death opens the way for possible preventive and curative measures.

Monocausality versus multicausality

Generally, analytical epidemiology deals with multicausality, i.e. disease is viewed as the resultant of a network of interdependent causes (network model of disease). In monocausal terms, alcoholism is caused by alcohol, like malaria by Plasmodium malariae/falciparum/vivax/ovale. In multicausal terms, however, malaria is brought about by predisposing factors such as poor hygiene, the presence of the Anopheles mosquito and lowered resistance to infection. Analogically in multicausal terms, alcoholism is brought about by predisposing and perpetuating factors leading to (excessive) alcohol consumption and alcoholism (cf. Chapter 4).

Interphenomenon relationships

Between two categories of phenomena the following types of relationships are possible:

a) *Independent relationship:* the two phenomena are *not* statistically correlated;
b) *Dependent relationship:* the two phenomena *are* statistically correlated;
   b)1. *Non-causal relationship:* statistical correlation between two phenomena based on their dependence on an identical third phenomenon;
   b)2. *Causal relationship;*
   b)3. *Circular-causal relationship:* statistical correlation between two phenomena based on the circumstance that both cause each other and both are consequences of each other.

Arguments for causal relationships in epidemiology

1) *Consistency investigation:* in various populations and under divergent conditions studies can be done to ascertain whether a statistical correlation between the distribution of a disease and the distribution of a suspected cause is maintained;
2) *Time sequence:* the order of succession always has to be the same: a change in the distribution of a disease has to be *preceded* by a change in the distribution of the suspected cause.

Construction of hypotheses

This can be done with the following methods:

a) *Method of difference:* two situations can be studied, in which there are two definitely different prevalences of a suspected cause;
b) *Method of similarity:* two divergent situations can be studied, in which there are definitely *similar* prevalences of a disease and concomitantly similar prevalences of one suspected cause;
c) *Method of parallel deviation:* this is a quantitative modification of a) and b);
d) *Method of analogy:* a disease with as yet unknown etiology, is compared with a disease with known etiology, under the given circumstance that in both diseases there is marked similarity as to the course of the disease.

Testing of hypotheses

This can be done in two ways:

1) *Retrospective investigation:* starting from the consequence (disease, death, health) the observer looks back at the preceding suspected cause(s).
2) *Prospective investigation:* the suspected cause is administered to a number of patients while another group of patients is given a non-causative innocuous agent, whereafter the distribution of disease is compared in both groups.

It is evident, that this study implies a *retrospective* investigation.

1.6. METHODS

Estimations of the prevalence of alcoholism in a certain region and in a certain period are often required for scientific and therapeutic or preventive purposes. A brief description is given of the following methods:

a) the population survey method;
b) the Jellinek estimation formula;
c) the estimation method based on data of mortality from alcoholism;
d) the estimation method based on data of mortality from suicides;
e) the Ledermann model of alcohol consumption.

1.6.1. The population survey method

This is a widely used method for the estimation of the prevalence

of alcoholism. Since the vast majority of typical clinical and laboratory findings in alcoholism are only to be found in drinking alcoholics or in alcoholics during the withdrawal syndrome, but *not* in sober alcoholics, up to now the most reliable information to characterize alcoholism even in sober alcoholics is to be found in examining the drinking history and in inquiring after frequent or regular occurrence of target symptoms in connection with alcohol consumption. This drinking history method furthermore has two advantages:

1) relatively low cost in comparison with survey methods which include extensive clinical and laboratory investigations;
2) the inquiry can be extended to include a number of variables to assess several medical, social and psychological hypotheses concerning alcoholism.

In this study the survey method was chosen as measuring instrument. To this end a questionnaire was designed, based on Jellinek's symptomatology (1960) and on the criteria presented by the Criteria Committee of N.C.A. (1972). The questions (variables) were roughly derived from four existing questionnaires:

1) The questionnaire by Gadourek (1963);
2) The questionnaire by Jones (1966);
3) The questionnaire of the University of Santiago, Chile, as mentioned by Horwitz et al. (1967);
4) The questionnaire by Bailey & Leach (1965).

The questionnaire used in this study was complemented by a modest number of questions, aimed at the assessment of some hypotheses, which on the basis of previous knowledge of certain aspects of drinking habits in Aruba, seemed to play an important role in specifically local drinking circumstances.

Also, on the basis of previous experience with approximately 250 alcoholics during a three years'period, some questions used in other studies were omitted, specifically those concerning aggressive or delinquent behavior while drinking and those concerning contacts with the police in connection with drunken behavior; the consistent experience with these questions was, that they often provoked reluctance and hence false negative responses.

Questions testing Jellinek's symptomatology (Jellinek, 1960) and the criteria from the Criteria Committee of N.C.A. (1972), were empirically found to discern most significantly between alcohol addicts,

non-addicted problem drinkers and social drinkers among our 250 alcoholic patients and non-alcoholic patients; it appeared, that most alcoholics did not seem to realize that specifically these symptoms marked them as alcohol-addicts, so that the probability of false negative responses could be considered as very close to nil. Consequently, the highest diagnostic level was assigned to this category of questions.

Some other questions, which also appeared to provoke reluctance and false negative responses, were those concerning the frequencies of intoxication and alcohol consumption, and whether or not one's drinking habits were ever accompanied by psychosis; consequently a relatively lower diagnostic level was assigned to these questions, in the differentiation between alcoholics, other pathological drinkers, social drinkers and abstainers.

Selzer (Moore, 1971; Selzer, 1971; Moore, 1972; Pokorny et al., 1972; Kaplan et al., 1974; Selzer et al., 1975; Zung & Charalampous, 1975) developed the Michigan Alcoholism Screening Test (MAST), consisting of $\pm$ 25 questions, including many symptoms from Jellinek's symptomatology. For scientific purposes such a test is preferable because of the fairly high repeatability of the results. The use of the MAST for the present study was not possible because the MAST came to the author's attention when this study's questionnaire was already devised. Moreover, it has the relative disadvantage that the same score is assigned to all its variables; a total score of $\geqslant$ 20% positive variables is considered as diagnostic, without taking into account any possibly critical symptom, such as for instance tolerance, withdrawal syndrome or loss-of-control, as a "conditio sine qua non" for the diagnosis; this mere summation of symptoms can be considered as incompatible with the definition of alcoholism as employed in this study, though it still might be compatible with the relatively broad definition of problem drinking.

This study's questionnaire was designed in two versions, questionnaire *A* and questionnaire *B*.

Questionnaire A, consisting of 61 questions, was presented to a random sample of the population of fifteen years and older (Drinking Age Population, D.A.P.) since alcoholism occurs only in those age groups, with as its principal purpose to detect the prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking,

social drinking, and abstinence; apart from that, a small number of hypotheses were to be tested by it. This questionnaire was completed by 708 respondents. Questionnaire A thus can be considered as a mainly *descriptive epidemiological survey*. This survey has been denominated "*survey A*".

Questionnaire B, consisting of 173 questions in which the original 61 questions of questionnaire A are included, was designed to be presented to a random sample of the D.A.P. as "control group" and to a non-random sample of patients with alcoholism. This is not a true "control group" because some alcoholics and other pathological drinkers were included. The main purpose of this questionnaire was to operatize certain concepts and by applying these to detect significant differences between alcoholics, other pathological drinkers, non-pathological drinkers and abstainers. This questionnaire was completed by 125 "alcoholics and other pathological drinkers" and by 99 "controls". Questionnaire B hence can be considered as both a *descriptive and analytical epidemiological survey*, with its purpose to detect significant differences between pathological drinking, non-pathological drinking and abstinence. The group of 99 "controls" who completed questionnaire B was denominated "*survey B*". The group of 125 "alcoholics and other pathological drinkers" who completed questionnaire B was denominated "*survey C*".

Survey A was performed in October and November 1972. Survey B and survey C were performed from October 1972 through January 1973, with a modest extension of survey B by 19 respondents in November 1973.

In addition to questionnaire B in this experimental and explanatory survey (survey B & survey C), one standard questionnaire was presented to smaller numbers of male "alcoholics" (survey C) and male "controls" (survey B): the *P.B.I.* (Parental Behavior Inventory).

The *P.B.I.* was added because of Van der Does de Willebois's (1965) statement, that *male* alcoholics generally had fathers with an "empty father's image" or an "unreachable father's image", while the Criteria Committee of N.C.A. (1972) makes mention of the role of the "rejecting but not punitive father". The P.B.I. consist of 72 questions and was designed by *Schaefer* in 1964 (Heydendael et al., 1972); it evaluates the respondent's perception of his or her father's (or mother's) behavior along a hypothetical model of parental beha-

vior devised on a set of two reciprocally perpendicular axes: rejection versus acceptance, and control versus autonomy.

1.6.2. The Jellinek estimation formula

Until recently, apart from the survey method, the most frequently used method to estimate the prevalence of alcoholism, was the one based on cirrhosis mortality data, developed by E. M. Jellinek (Jellinek, 1951, 1959). This so-called "Jellinek estimation formula" is easily applicable. It requires but one basic information, i.e. the number of male and female deaths from liver cirrhosis in a given year. It can be calculated for separate countries and regions, and the various resulting prevalence rates can be compared.

1.6.3. The estimation method based on data of mortality from alcoholism

Schmidt & De Lint (1970) have proposed and used the following formula:

$$A = \frac{D_a}{R_a} \times 10{,}000$$

A = the total number of alcoholics in a given year.
D_a = the total number of deaths from alcoholism in a given year.
R_a = the average number of alcoholics yearly dying from alcoholism per 10,000 alcoholics; this proportion can be determined in longitudinal studies over a number of years.

Mortality from alcoholism implies all deaths caused by alcoholismic syndromes such as delirium tremens, lethal alcohol-intoxication, alcoholic convulsions, and those caused entirely or partially by alcohol such as gastrointestinal hemorrhage due to alcohol.

1.6.4. The estimation method based on data of mortality from suicides

Schmidt & De Lint (1970) have proposed and used the following formula:

$$A = \frac{P_s \times D_s}{R_s} \times 10{,}000$$

A = the total number of alcoholics in a given year.
D_s = the total number of suicides in a given year.
P_s = the percentage of D_s due to alcoholism.

R_s = the average number of alcoholics dying from suicide per annum per 10,000 alcoholics; P_s and R_s can both be determined in longitudinal studies over a number of years.

1.6.5. The Ledermann model of alcohol consumption

Another estimation method was developed by Ledermann (1956, 1964), based on average annual alcohol consumption per drinker in a given population. This method has been applied several times for a number of countries (De Lint, 1968, 1974, 1975; De Lint & Schmidt, 1968, 1970, 1971, 1971a, 1971c; Schmidt & De Lint, 1970).

This method is known as the logarithmic-normal distribution of alcohol consumption, the Ledermann model of alcohol consumption, the Ledermann equation, the Ledermann formula.

De Lint et al. (1970) found a positive correlation between prevalence rates of alcoholism calculated by the Jellinek estimation formula and by the Ledermann equation, cf. *tab. 1.1.*

Table 1.1.

Correlations between liver-cirrhosis mortality and annual per capita alcohol consumption

Country	Series	Correlation-coefficient	Probability
Australia	1938 - 1959	0 65	⟨0 005
Belgium	1929 - 1959 (without 1940 - 1945)	0 75	⟨0 001
Canada	1927 - 1960	0 88	⟨0 001
Canada	9 provinces 1955	0 81	⟨0 01
Finland	1933 - 1957	0 78	⟨0 001
France	1925 - 1958	0 62	⟨0 001
France	23 departments 1950	0 76	⟨0 001
The Netherlands	1927 - 1958	0 57	⟨0 001
Sweden	1926 - 1956	0 45	⟨0 05
U S A	46 states 1957	0 86	⟨0 001

Note Liver-cirrhosis mortality in all series is expressed as the number of deaths per 100,000 inhabitants of 20 years or older

De Lint & Schmidt (De Lint & Schmidt, 1968; Schmidt & De Lint, 1970) in a study in the Canadian province of Ontario also found a

positive correlation between prevalence estimations by the Ledermann equation and by the modified Jellinek formula as proposed by Popham (1956) on the one hand, and prevalence estimations based on data from the mortality from suicide and from alcoholism (Manual of statistic classification of diseases, injuries and causes of death, 1955). The reliability of estimations of alcoholism prevalence by the Ledermann equation would be augmented if reasonable concordance is obtained with estimates based on mortality data (Schmidt & De Lint, 1970).

1.6.6. Factor analysis

Factor analysis is a method to reduce an extensive number of variables to a small number on the basis of the intercorrelations between these variables. It allows one to mathematically define how certain variables cluster together, based on their intercorrelations (Wanberg & Knapp, 1970). A *factor* is interpreted as a pattern of characteristics which can be assessed to have a common basis. A *factor,* is a mathematically based and objective representation of what is more generally understood as a symptom cluster or trait. If drinking symptoms or behavior tend to form different clusters or factors and if these are orthogonal in nature, it can be said that they are independent of one another. If one factor is independent of another, a respondent's score on the one cannot be predicted by his scores on the other (Wanberg & Knapp, 1970).

In this study, factor analysis is employed for the following purposes:

1) To assess the postulated *unidimensional model of alcoholism* (Jellinek, 1952, 1960) versus the multidimensional model (Wanberg, 1969; Wanberg & Knapp, 1970); this is described in Chapter 2, and involves a descriptive epidemiological approach;
2) To assess the postulated *unidimensional model of problem drinking* versus the multidimensional model; this is also described in Chapter 2, and involves a descriptive epidemiological approach;
3) To assess the specific dimensions of a number of grouped variables correlated with alcoholism in a causal (or circular causal) relationship; this is described in Chapter 5, and involves an analytical epidemiological approach.

1.7. DESCRIPTION OF SURVEY A AND SURVEY B SAMPLES

a) *Survey A sample.* With the aid of the Citograf every fourtieth name was drawn from the population register of the Census Department in August 1972. By this procedure a total of 1,528 names was drawn. From this random sample all persons younger than fifteen years were removed, since alcoholism only occurs in those of fifteen years and older. Thus a number of 1,041 names remained, all belonging to the D.A.P.
In order to be able to extend this sample in case of too large a proportion of refusers and other dropouts, from this total of 1,041 names every fourth name was removed, so that a workable sample of 779 names was left, to be used as *survey A.*

b) *Survey B sample.* For survey B the procedure was as follows: out of the previously mentioned total of 1,041 every tenth name was selected. These 104 names were originally used as *survey B* sample.

Both survey A and survey B turned out to have such a large proportion of refusers and other drop-outs, that these samples had to be extended.

Because of the very high proportion of refusers and other drop-outs in *survey B,* this sample was extended by a number of *male* respondents by choosing every fifth name from the previously mentioned total of 779 names in survey A sample removing the thus selected female respondents; only male respondents were chosen for this extension of the survey B sample, because the vast majority of alcoholics in survey C, with whom survey B respondents had to be compared, were men, and also because there appeared to be too large a discrepancy between the totals of P.B.I. questionnaires in survey B and in survey C. After subtracting the total of refusers and other dropouts a definite total of 99 "controls" in the survey B sample remained.

Survey A ultimately yielded a total of 708 respondents (approximately 2% of the D.A.P.). In survey A there was a starting total of 876 respondents, of which *168 (19.1%)* dropped out for the following reasons:

1)	Refusers	25
2)	Migration to other countries	54
3)	Untraceable migration within the island	32
4)	On vacation	18
5)	Mental retardation, dementia, deaf-muteness, psychosis	12
6)	Untraceable; never at home	25
7)	Death	2
	(Total)	168

The total of 779 names in survey A was extended to 876 as follows: After 400 respondents had been approached and/or interviewed, it appeared that 81 were dropouts (20%). Hence it was decided to extend with a total of 97 (= 81 + 20% of 81). These 97 names were chosen aselectly from the remaining 262 (= 1,041 — 779) in the D.A.P. sample mentioned above, while district and sex were kept constant for every respondent to be chosen to replace a dropout.

In survey B there was a starting total number of 141 respondents, of which *42 (29.8%)* dropped out for the following reasons:

1)	Refusers	10
2)	Migration to other countries	10
3)	Untraceable migration within the island	6
4)	Untraceable; never at home	12
5)	Mental retardation, dementia, deaf-muteness, psychosis	4
	(Total)	42

The strong impression exists, that "untraceable; never at home" respondents mainly represent refusers too. In survey A both groups ("refusers" and "untraceable") represent *5.7%* of the total of 876 respondents. In survey B these two groups constitute *15.6%* of the total of 141 respondents. Hence it is evident, that the proportion of refusers was nearly three times greater in survey B than in survey A. The most plausible explanation for this marked difference is the difference in duration between both questionnaires: questionnaire A took an average of 20 minutes per respondent, while questionnaire B (plus P.B.I.) took an average of 90 minutes per respondent. It is easily understandable, that people would tend to refuse to answer a very lengthy questionnaire rather than a short one. An additional

explanation: presumably a significant proportion of "dropouts" in survey A and survey B may be pathological drinkers since it is well known that alcoholics commonly are characterized by denial of any problems.

A population pyramid was drawn for survey A sample and compared with a population pyramid for the population of fifteen years and older (D.A.P.), based on official data from the 1972 census (Eerste algemene volks- en woningtelling Nederlandse Antillen, 1972), cf. *fig. 1.4.*

A fairly close concordance was found between both population pyramids. Hence survey A sample can be considered as a reliably drawn sample of the D.A.P.

figure 1.4.

Survey A sample compared with the population of Aruba according to the 1972 census.

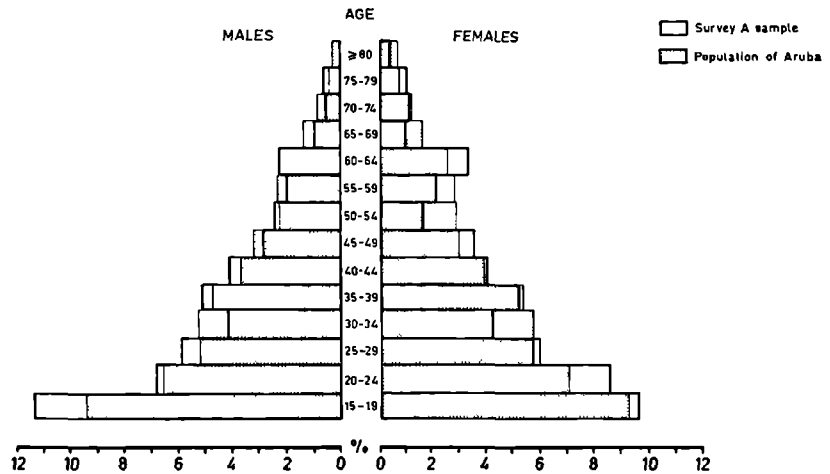


Table 1.2. gives a comparison between the distribution of survey A sample and the total population of the island over the six districts.

There is reasonable concordance between survey A sample and the total population as regards the distribution in the six districts.

Table 1.2.

Distribution of survey A sample as compared to the total population

	Survey A sample			Total population*	
District	Observed	Percentage	Expected	Total	Percentage
Oranjestad	204	28.8%	194	16.711	27.5%
Nort	75	10.6%	77	6.652	10.9%
Paradera	51	7.2%	55	4.695	7.7%
Santa Cruz	101	14.3%	101	8.715	14.3%
Sabaneta & Brazil	104	14.7%	95	8.126	13.4%
San Nicolás	173	24.4%	186	15.901	26.2%
(Total)	708	100.0%	708	60.800	100.0%

* Unofficial data from the Census Department, 1972.

$\chi^2 = 2.52$
df = 5
$p > 0.05$ (not significant)

There is reasonable concordance between survey A sample and the total population as regards the distribution in the six districts.

1.8. INTERVIEWERS

Twenty-one interviewers participated in the three surveys, including the author who only participated in survey C ("alcoholics"). Of these 21 interviewers 14 were female, and 7 were male. Most of them were chosen because of their previous experience with interviewing in other surveys. Most of them were social workers, nurses, or other paramedical workers. A review of their professions is presented in *table 1.3.*:

Table 1.3.

Interviewers

	Male	*Female*
Social workers	1	7
Nurses	2	5
Government officials	2	1
Students	1	0
Teachers	0	1
Physicians	1	0
(Total)	7	14

They all received a briefing in August 1972 on the aims of the survey and the significance and interpretation of the questions in questionnaire A; this briefing was repeated twice, to give the interviewers the opportunity to ask questions regarding questionnaire A. Five interviewers received a briefing in October 1972 on the significance and interpretation of questions in questionnaire B, and the P.B.I. The author only participated in survey C as an interviewer because of his time mainly being consumed by daily medical practice and the organization of surveys A and B.

Chapter 2

DESCRIPTIVE EPIDEMIOLOGY: ESTIMATION OF THE PREVALENCES OF ALCOHOLISM, GAMMA-PREALCOHOLISM, PREALCOHOLISM, PROBLEM DRINKING, SOCIAL DRINKING AND ABSTINENCE

2.1. METHODS

The following methods are employed in this study:

1) *The population survey method;*
2) *The Jellinek estimation formula;*
3) *The Ledermann equation.*

2.1.1. The population survey method

Critical questions to detect the prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking and abstinence, mainly derived from Jellinek's symptomatology, were used in both questionnaire A and in questionnaire B (cf. *Appendix A*).

Questionnaire A was completed by 708 respondents, a random sample of the Aruban population of fifteen years and older.

Questionnaire B was completed by 99 respondents from the original random sample of 708 respondents as a "control group", and by 125 non-randomly selected patients with alcoholism, (gamma-) prealcoholism and problem drinking.

2.1.1.1. Factor analysis for the dimensions: alcoholism and problem drinking.

Before computer-detection of the six diagnostic categories (alcoholism; gamma-prealcoholism; prealcoholism; problem drinking; social drinking; abstinence) was performed, *factor-analysis* was executed among:

a) those questions (= variables) considered diagnostic for the diagnosis *alcoholism;*
b) those questions (= variables) considered diagnostic for the diagnosis *problem drinking.*

Factor analysis was described in Chapter 1. This factor-analysis was executed in the *combined* population of 125 "pathological drinkers" and 99 "controls" who completed questionnaire B.

In concordance with this study's broader definition of problem drinking (cf. Chapter 1), the variables considered diagnostic for the diagnosis alcoholism were also included in the set of variables on which factor-analysis for the dimension "problem drinking" was to be executed.

Before factor-analysis was performed, a tentative score was given to every possible response for each variable; these scores were weighed according to the diagnostic levels assigned to them by the Criteria Committee of N.C.A. (1972) or by the American Psychiatric Association (1968).

Table 2.1. shows factor analysis for the dimension "alcoholism", all variables included therein, and factor loadings per variable; variables are numbered as in Appendix A.

Table 2.2. shows factor analysis for the dimension "problem drinking", all variables included therein, and factor loadings per variable; variables are numbered as in Appendix A.

Table 2.1.

Factor analysis for the dimension "alcoholism", factor I; extracted variance 0.4415.

Variable No.	Content of variable	Factor loading
64	Physical dependence	0.8768
37	Frequency of intoxication	0.8070
62	Loss-of-control	0.8039
72	Delirium tremens	0.7573
71	Alcoholic hallucinosis	0.7379
36	Frequency of alcohol consumption	0.6867
66	Early morning drink	0.6636
58	Psychological dependence	0.6491
69	Alcoholic gastritis	0.6090
168	Craving	0.6012
30	Beverage of choice	0.5969
70	Alcoholic polyneuropathy	0.5645
67	Blackout	0.5579
73	Alcoholic convulsions	0.5045
74	Alcoholic psychosis	0.3640

Two other factors were extracted in this factor analysis, factor II (extracted variance 0.0797) and factor III (extracted variance 0.0670).

Factor I was accepted, since all except one variable had a factor loading $\geq$ 0.4; this variable was still included since its loading was but a little lower than 0.4, namely 0.3640 (alcoholic psychosis). Generally only factor loadings $\geq$ 0.4 or $\leq$ -0.4 have been accepted.

Factor II was *rejected,* since it contained only four variables with factor loadings $\geq$ 0.4 or $\leq$ -0.4.

Factor III was *rejected,* since it contained only one variable (alcoholic psychosis) with a factor loading $\geq$ 0.4.

Total extracted variance was 0.5882, of which 0.4415 by factor I.

Two variables were excluded from the set of variables considered as diagnostic for the diagnosis "alcoholism":

a) *Frequency of drinking* (factor loading 0.6867).
   Reason: social drinkers are able to consume alcoholic beverages from "(less than) once a year" to "every day".
b) *Beverage of choice* (factor loading 0.5969).
   Reason: social drinkers can have as their beverage of choice both soft-drinks and alcoholic beverages (beer, wine, or distilled spirits).

The variable no. 74 ("alcoholic psychosis") is retained notwithstanding its low factor loading (0.2815), while generally only levels $\geq$ 0.4 have been accepted.

Four other factors were extracted: factor II (variance 0.0507); factor III (variance 0.0443); factor IV (variance 0.0385); factor V (variance 0.0337).

Factor I was accepted, since all except two variables had a factor loading $\geq$ 0.4.

Factors II, III, IV and V were rejected, since they had only three, two, zero or two factor loadings $\geq$ 0.4.

Total extracted variance was 0.6357, of which 0.4684 by factor I.

As shown in *table 2.1.* and *table 2.2.* the *unidimensional models* for alcoholism and *problem drinking* (Jellinek, 1952, 1960) are clearly confirmed by factor analysis, while the *multidimensional models,* as found by Wanberg et al. through factor analysis, are rejected (Wanberg, 1969; Wanberg & Knapp, 1970). Wanberg et al. stated,

Table 2 2

Factor analysis for the dimension "problem drinking", factor I, extracted variance 0 4684

Variable No	Content of variable	Factor loading
150	Amount of help received from authorities for drinking problem	0 9321
76	Location of treatment for drinking problem	0 9234
146	Authorities consulted for drinking problem	0 9155
173	Frequency of intoxication after treatment	0 9121
75	Kind of treatment for drinking problem	0 9097
166	Frequency of alcohol consumption after treatment	0 9044
167	Intensity of problems after treatment	0 9012
154	Frequency of attending AA-meetings	0 8612
151	AA-membership	0 8585
64	Physical dependence	0 8348
62	Loss-of-control	0 8102
37	Frequency of intoxication	0 7978
158	Self-imposed abstinence to "prove" control over alcohol	0 6845
161	Financial troubles because of drinking	0 6638
33	Preferred drinking companions	0 6496
58	Psychological dependence	0 6476
72	Delirium tremens	0 6473
147	Established diagnosis alcoholism or problem drinking	0 6386
66	Early morning drink	0 6319
36	Frequency of alcohol consumption	0 6255
160	Trouble at work because of drinking	0 6195
164	Unhappy family-life because of drinking	0 6027
71	Alcoholic hallucinosis	0 5983
159	Quarrels or fights because of drinking	0 5893
30	Beverage of choice	0 5825
168	Craving	0 5703
70	Alcoholic polyneuropathy	0 5366
67	Blackout	0 5243
152	Frequency of speeches at AA-meetings	0 5193
69	Alcoholic gastritis	0 4991
73	Alcoholic convulsions	0 4621
162	Familial ostracism because of drinking	0 4295
32	Preferred location for drinking	0 4183
163	Longing for alcohol daily at same time	0 4149
165	Solitary drinking	0 3946
74	Alcoholic psychosis	0 2815

that there were *several* dimensions of alcoholism to be found among *1891* patients admitted to their Alcoholism Division at Fort Logan Mental Health Center, and that those dimensions were statistically independent rather than being related on a continuum from early through middle to late stage alcoholism; hence they concluded that the unidimensional model (Jellinek, 1952, 1960) failed to explain the phenomenon of alcoholism (Wanberg & Knapp, 1970). The findings in this study are at variance with those mentioned by Wanberg et al. (Wanberg, 1969; Wanberg & Knapp, 1970). It is possible, that this study's rejection of the postulated multidimensional model is due to:

1) the fact that this study deals with a much *smaller* sample (28 abstainers; 41 social drinkers; 24 problem drinkers and 131 gammaprealcoholics & alcoholics) compared to the 1891 pathological drinkers in the study by Wanberg et al.;
2) the fact that factor analysis in this study was performed in a combined population of abstainers, social drinkers and pathological drinkers, while in Wanberg's study this was done only in pathological drinkers.

This rejection is certainly not explicable by differences in interview questions, since the variables used as diagnostic criteria in Wanberg's study are practically the same as those in this study.

2.1.1.2. Criteria for the diagnoses: alcoholism, gamma-prealcoholism, prealcoholism, problem drinking, social drinking and abstinence.

Table 2.3. shows the criteria used for the diagnosis "alcoholism".
Table 2.4. shows the criteria used for the diagnosis "gamma-prealcoholism".
Table 2.5. shows the criteria used for the diagnosis "prealcoholism".
Table 2.6. shows the criteria used for the diagnosis "problem drinking".
Table 2.7. shows the criteria used for the diagnosis "abstinence".

It is obvious, that "*social drinking*" remains as diagnostic category by the exclusion of the five other categories.

Table 2 3

Criteria for the diagnosis "alcoholism"

Variable No	Content of variable	Scores
	A) *On one variable*	
64	Physical dependence	2
or 71	Alcoholic hallucinosis	2
or 72	Delirium tremens	2
	B) *On two variables*	
64	Physical dependence (unconvincing)	1
or 73	Alcoholic convulsions	1
	To which is added one of the following variables	
37	Frequency of intoxication	$\geqslant$ 4
58	Psychological dependence	1
62	Loss-of-control	1
66	Early morning drink	1
67	Blackout	1
69	Alcoholic gastritis	1
70	Alcoholic polyneuropathy	1
73	Alcoholic convulsions	1
74	Alcoholic psychosis	1
168	Craving	1

Table 2 4

Criteria for the diagnosis "gamma-prealcoholism"

Variable No	Content of variable	Scores
62	Loss-of-control	1
	If the following variables have score 0	
64	Physical dependence	0
71	Alcoholic hallucinosis	0
72	Delirium tremens	0
73	Alcoholic convulsions	0

Table 2 5

Criteria for the diagnosis "prealcoholism"

Variable No	Content of variable	Scores
64	Physical dependence (unconvincing)	1
or 73	Alcoholic convulsions	1

	If the following variables have score 0	
62	Loss-of-control	0
71	Alcoholic hallucinosis	0
72	Delirium tremens	0
58	Psychological dependence	0
66	Early morning drink	0
67	Blackout	0
69	Alcoholic gastritis	0
70	Alcoholic polyneuropathy	0
74	Alcoholic psychosis	0
168	Craving	0
	And if the following variable has score <4	
37	Frequency of intoxication	< 4

Table 26

Criteria for the diagnosis "Problem drinking"

Variable No	Content of variable	Scores
{ 32	Preferred location for drinking	2 } = 5
{ 33	Preferred drinking companions	+ 3 } = 5
or 37	Frequency of intoxication	$\geq$ 2
or 58	Psychological dependence	1
or 66	Early morning drink	1
or 67	Blackout	1
or 69	Alcoholic gastritis	1
or 70	Alcoholic polyneuropathy	1
or 74	Alcoholic psychosis	1
or 75	Kind of treatment for drinking problems	1
or 76	Location of treatment for drinking problems	1
or 146	Authorities consulted for drinking problems	1
or 147	Established diagnosis alcoholism/ problem drinking	1
or 150	Amount of help received from authorities	1
or 151	AA-membership	1
or 152	Frequency of speeches at AA-meetings	1
or 154	Frequency of attending AA-meetings	1

	158	Self-imposed abstinence to prove control over alcohol	1	
	159	Quarrels or fights because of drinking	+ 1	
	160	Trouble at work because of drinking	+ 1	
or:	161	Financial troubles because of drinking	+ 1	⩾ 5
	162	Familial ostracism because of drinking	+ 1	
	163	Longing for alcohol daily at same time	+ 1	
	164	Unhappy family-life because of drinking	+ 1	
	165	Solitary drinking	+ 1	
or:	166	Frequency of drinking after treatment	1	
or:	167	Intensity of problems after treatment	1	
or:	168	Craving	1	
or:	173	Frequency of intoxication after treatment	1	
		If the following variables have score 0:		
	62	Loss-of-control	0	
	64	Physical dependence	0	
	71	Alcoholic hallucinosis	0	
	72	Delirium tremens	0	
	73	Alcoholic convulsions	0	

Table 2.7.

Criteria for the diagnosis: "abstinence".

Variable No.	Content of variable	Scores
36	Frequency of drinking	0
37	Frequency of intoxication	0
30	Beverage of choice	0
	Only if the respondent does *not* respond to the diagnoses: alcoholism, gamma-prealcoholism, prealcoholism or problem drinking.	

2.1.1.3. Estimation of the prevalences of the six diagnostic categories.

This was performed on the basis of the criteria mentioned in tables 2.3. through 2.7., whilst the diagnostic category "social drinking" remains by the exclusion of the five other diagnoses.

Table 2.8. shows the prevalences of the six diagnostic categories in the original random sample of 708 respondents from the D.A.P. (Drinking Age Population, i.e. the population of fifteen years and older), and the corresponding prevalences in the real D.A.P. according to the findings in this study, in absolute numbers.

Table 2 8

Prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking, social drinking and abstinence in Aruba, 1972

Diagnostic category	Drinking Age Population (D A P) sample (N = 708)	Percentages in D A P sample	Absolute numbers in D A P. (N = 35,133)	95% - Confidence interval*
Abstinence	181	25 6	8,994	7,868 — 10,120
Social Drinking	371	52 4	18,409	17,114 — 19,704
Problem Drinking	83	11 7	4,111	3,272 — 4,950
Prealcoholism	6	0 8	281	48 — 514
Gamma-pre-alcoholism	16	2 3	808	421 — 1,195
Alcoholism	51	7 2	2,530	1,860 — 3,200
Total	708	100 0	35,133	———

* 95%-Confidence interval indicates, that there is a probability of 95% that these figures are between the upper and lower limits as mentioned

As can be seen in *Table 2.8.* the "Drinking Population" (D.P.) amounts to *74.4%* of the D.A.P., that is 26,139 in absolute numbers. The D.P. is composed of the D.A.P. *minus* the proportion of *abstainers* in the D.A.P. It can be concluded, that though average annual consumption of absolute alcohol per capita of the D.P. and hence the prevalence of alcoholism in Aruba is higher than in the Netherlands for example, this certainly is *not* due to a higher prevalence of drinkers; on the contrary, according to Gadourek (Gadourek, 1963) in the late nineteen-fifties the Netherlands had a higher prevalence of drinkers, i.e. 82.1% (among respondents of 20 years and older).

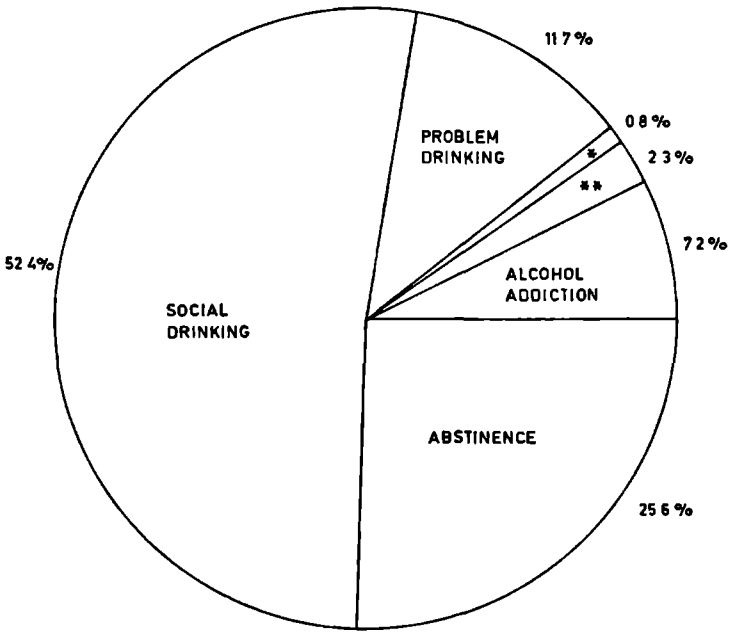
Abstinence occurs with a higher prevalence (25.6%) in Aruba than in the Netherlands (17.9%) (Gadourek, 1963).

Fig. 2.1. depicts the data of table 2.8.

The prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking and social drinking in the *D.P.* (= D.A.P. minus abstainers) are shown in *table 2.9.*

figure 2 1

PREVALENCES OF ALCOHOLISM, GAMMA-PREALCOHOLISM, PREALCOHOLISM, PROBLEM DRINKING, SOCIAL DRINKING AND ABSTINENCE, ARUBA, 1972



* PREALCOHOLISM, ** GAMMA-PREALCOHOLISM

Table 2 9

Prevalences of alcoholism, gamma prealcoholism, prealcoholism, problem drinking and social drinking in the drinking population (D P) Aruba, 1972

Diagnostic category	Drinking Population (D P) Sample	Percentage in D P Sample
Social Drinking	371	70 4
Problem Drinking	83	15 8
Prealcoholism	6	1 1
Gamma-prealcoholism	16	3 0
Alcoholism	51	9 7
Total	527	100 0

It may be concluded from *table 2.9.* that among all *drinkers* in Aruba, 29.6% have entered the "alcoholismic learning process" (Keller, 1972) and hence are in danger of developing full-blown alcoholism and its harmful complications such as liver cirrhosis.

The prevalences of alcoholism, (gamma-)prealcoholism and problem drinking among the 125 non-randomly selected patients with "pathological drinking" ("survey C") are shown in *table 2.10.*

Table 2.10

Prevalences of alcoholism, gamma-prealcoholism, prealcoholism and problem drinking in a non-random sample of 125 "pathological drinkers", Aruba, 1972.

Diagnostic category	Absolute Numbers	Percentages in survey C sample
Problem Drinking	4	3.2
Prealcoholism	0	0.0
Gamma-prealcoholism	4	3.2
Alcoholism	117	93.6
Total	125	100.0

It may be concluded that the diagnosis "*alcoholism*", as previously hypothesized in this sample of 125, was only applicable to 93.6% of these patients, and hence that in 6.4% this diagnosis was *not* correct.

The prevalences of alcoholism, gamma-prealcoholism, problem drinking, social drinking and abstinence in a random sample of 99 respondents from the D.A.P. ("survey B") *plus* the above mentioned non-random sample of 125 "pathological drinkers ("survey C") are shown in *table 2.11.*

Table 2.11

Prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking, social drinking and abstinence in the combined population of a random sample of 99 respondents from the D.A.P. plus a non-random sample of 125 "pathological drinkers", Aruba, 1972

Diagnostic category	Absolute Numbers	Percentages in survey B & C sample
Abstinence	28	12.5
Social Drinking	41	18.4
Problem Drinking	24	10.7
Prealcoholism	0	0.0
Gamma-prealcoholism	5	2.2
Alcoholism	126	56.2
Total	224	100.0

This combined population, as mentioned in *table 2.11.*, is used in Chapters 5 & 6 to test the various theories concerning the etiology of alcoholism.

2.1.2. The Jellinek estimation formula

2.1.2.1. Description of the Jellinek formula.

Until recent years, apart from the population survey method, the most frequently employed estimation method to determine the prevalence of alcoholism was the Jellinek estimation formula, based on data of mortality from liver cirrhosis and alcoholic liver cirrhosis (Jellinek, 1951, 1959; Keller & Efron, 1955; Popham, 1956; Brenner, 1959; Argeriou, 1974). The Jellinek estimation formula is easily applicable; it requires a single datum, namely the number of (male and female) deaths due to liver cirrhosis, it can be applied for separate countries and regions, and the thus found prevalence rates can be compared for the regions studied.

The Jellinek estimation formula is as follows

$$A = \frac{P_c \times D_c}{K_c} \times R$$

A = the total number of living alcoholics in a given year, this is the unknown value which is to be determined
D_c = the recorded number of deaths due to liver cirrhosis from all causes in a given year
P_c = the percentage of all deaths due to liver cirrhosis, which can be ascribed to alcoholism.

From an accurate trend-analysis of mortality data in the U S A over the period 1900-1945, Jellinek (Jellinek, 1951) concluded that P_c was a constant, with a value of 62 8% for males and 21 6% for females, i e 42 2% for both sexes

The trend of general mortality in the U S A over the period 1910-1940 was compared with the trend of the mortality due to liver cirrhosis The period 1915-1932 was the so-called Prohibition Era, during which production and sales of alcoholic beverages were gradually completely forbidden over the whole area of the U S A

In the period 1910-1915 liver cirrhosis mortality decreased at the same rate as general mortality

In the period 1916-1920 liver cirrhosis mortality, as well as tuberculosis mortality, decreased at a much higher rate than general mortality, while in this period general mortality decreased with 7 1%, liver cirrhosis mortality decreased with 42 2% The difference between these two percentages, approx 35%, was interpreted as the proportion (P_c) of liver cirrhosis mortality ascribable to alcohol

During the period 1933-1940 tuberculosis mortality further decreased, while liver cirrhosis mortality increased to approximately the same values as before the Prohibition Era.

Later on, the trend-analysis was extended to the period 1900-1945, the value of P_c was then revised at 51 5% for males and 17 7% for females, with a mean of approx 40% for both sexes

K_c = the percentage of all "alcoholics-with-complications" dying from liver cirrhosis

This factor also appears to be a constant The value of K_c was determined by Jellinek from a study of obductions of approx 100,000 ' alcoholics with-complications", of this total of 100,000 approx 9,000 had liver cirrhosis, while 7 71% of these 9,000 had *died* from liver cirrhosis, hence (7 71% x 9% = *0 694%*) represents the percentage of all "alcoholics with-complications" dying from liver cirrhosis

R = the ratio $\frac{\text{all alcoholics}}{\text{alcoholics-with-complications}}$

Jellinek's original analysis of data from archives from various American clinics for alcoholism, detected that only 25% of all alcoholics had somatic or psychiatric complications, hence R was initially valued at *4* A later analysis concluded that the proportion of ' alcoholics-with-complications" in the U S A had dropped from 25% to 18 75% so that the value of R was revised at *5 3* For the U S A R was initially estimated at 4, but later at 5 3 (Jellinek, 1951, 1959), because the proportion of "alcoholics-with-complications ' had decreased between 1942 and 1959 by obligatory multivitaminization of bread the prevalence of alcoholic polyneuropathy in the U S A had decreased considerably (Jellinek, 1959)

Nevertheless the value of R appears to differ from country to country approx 4 in England, Finland, Norway, Denmark, Sweden and Canada, approx 2 in France and Australia, approx $1^1{}_2$ in Switzerland (Keller & Efron, 1955)

The following considerations have played a role when Jellinek (Jellinek, 1951) devised his estimation formula

a) The treatment of liver cirrhosis has not undergone radical changes in the first half of the twentieth century, and hence trends in liver cirrhosis mortality do *not* reflect trends in *treatment* of liver cirrhosis
b) Under reporting of liver cirrhosis mortality does not seem to be probable, as might well be the case with data of mortality from alcoholism, in view of many physicians' negative attitudes towards this diagnosis
c) When making the diagnosis "death from liver cirrhosis", most physicians will probably use more or less the same criteria

Popham (Popham, 1956) has proposed a modification of the Jellinek estimation formula This modification implies the substitution of K_c and R by a single factor R_c, representing the percentage of *all* alcoholics (*with* and *without* complications) dying from liver cirrhosis

The Popham-modification of the Jellinek formula is as follows

$$A = \frac{P_c \times D_c}{R_c}$$

In this formula $R_c = \frac{K_c}{R}$, as compared to the original factors K_c and R in the Jellinek formula If K_c and R are known in a population, then the factor R_c is applicable too For Canada R_c is valued at 0 001653 (Schmidt & De Lint, 1970)

2.1.2.2. Application of the Jellinek formula; Aruba, 1961-1970.

For the application of the Jellinek formula in countries outside the U.S.A. it is important to determine the specific value of P_c in certain periods (Popham, 1956). In the period 1961-1970 (ten years) there have been 40 deaths from liver cirrhosis in Aruba, and 21 of these were due to alcoholic liver cirrhosis, so that P_c is valued at *52.5%*.

In the period 1961-1965 (five years) of the 14 deaths from liver cirrhosis, 8 were due to alcoholic liver cirrhosis, i.e. P_c = *57%*.

In the period 1966-1970 (five years) of the 26 deaths from liver cirrhosis, thirteen were due to alcoholic liver cirrhosis, i.e. P_c = *50%*.

Table 2.12

Deaths from liver cirrhosis and alcoholic liver cirrhosis, Aruba, 1961-1970

Year	Deaths from liver cirrhosis due to alcoholism	Deaths from liver cirrhosis due to other causes	Deaths from liver cirrhosis, cause of liver cirrhosis unknown
1961	4	2	—
1962	2	—	—
1963	—	—	1
1964	—	—	—
1965	2	3	1
1966	2	1	—
1967	2	—	—
1968	1	4	—
1969	2	4	—
1970	6	3	—
Total	21	17	2

Table 2.12. shows the annual numbers of deaths from liver cirrhosis due to alcoholism and due to other causes. In 1963 and 1964 no deaths by liver cirrhosis have been registered, and in 1962 and 1967 no deaths from alcoholic liver cirrhosis. The relatively low numbers in *table 2.12.* are a reflection of the relatively small total island population, which increased from approx. 57,000 in 1961 to approx. 61,000 in 1970.

These relatively low figures, as well as the absence of deaths from (alcoholic) liver cirrhosis in a number of years, make it impossible to apply the Jellinek formula for *each* year in the 1961-1970 period.

Instead, the Jellinek estimation formula is applied for the two five-year-periods 1961-1965 and 1966-1970, and for the decade 1961-1970. Since it can be expected from a computation over a five-year-period, that the number of alcoholics will be 5 times greater than in a computation over a one-year-period, the number of A for each each year in that five-year-period) will be multiplied by $^1/_5$. For the five-year-period (representing the *mean* number of alcoholics for decade 1961-1970 consequently this implies a multiplication by $^1/_{10}$.

These computations are shown in *table 2.13.* These computations are given with two values of R, namely:

a) R = 5.3 (Jellinek, 1959).

b) R = 3.5 This is the mean value of R in the following 10 countries: the U.S.A., Great Britain, Finland, Norway, Denmark, Sweden, Canada, France, Australia and Switzerland (Keller & Efron, 1955).

Table 2.13

Application of the Jellinek estimation formula, Aruba, 1961-1970.

Period	Jellinek formula	R=5.3	R=3.5
	$A = \frac{P_t \times D_t}{K_t} \times {}^1/_5$ or $^1/_{10} \times R$		
1) 1961-1965	$A = \frac{0.57 \times 14}{0.00694} \times {}^1/_5 \times (5.3 \text{ or } 3.5) =$	1,219	807
2) 1966-1970	$A = \frac{0.50 \times 26}{0.00694} \times {}^1/_5 \times (5.3 \text{ or } 3.5) =$	1,982	1,311
3) 1961-1970	$A = \frac{0.525 \times 40}{0.00694} \times {}^1/_{10} \times (5.3 \text{ or } 3.5) =$	1,603	1,059

2.1.3. The Ledermann equation

2.1.3.1. Description of the Ledermann equation.

In the nineteen-fifties an estimation method was developed by Ledermann, based on mean annual consumption of absolute alcohol (100 vol.%) per capita of the drinking population in a certain population (Ledermann, 1956, 1964). Later, this formula has been repeatedly applied for several countries (De Lint, 1968, 1974, 1975; De Lint & Schmidt, 1968, 1970, 1971a, 1971c; Schmidt & De Lint, 1970).

This method is known as the logarithmic-normal distribution of alcohol consumption, the Ledermann model of alcohol consumption or the Ledermann formula. Schmidt et al. (Schmidt & De Lint, 1970; De Lint & Schmidt, 1970, 1971) have found a positive correlation between estimations of alcoholism prevalence rates with the Jellinek formula and the Ledermann equation (cf. *table 1.1.* in Chapter 1).

In a study in the Canadian province Ontario (Schmidt & De Lint, 1970) a positive correlation was found between estimations of alcoholism prevalence rates with the Ledermann equation, the Popham-modification of the Jellinek formula, an estimation method based on figures of mortality due to alcoholism and an estimation method based on figures of mortality due to suicides (Manual of statistic classification of diseases, injuries and causes of death; 1955). Reliability of estimations of alcoholism prevalence based on annual alcohol consumption could be enhanced, if reasonable concordance could be obtained with estimations based on mortality data (Schmidt & De Lint, 1970).

For further understanding the implications of the Ledermann equation it is important to be aware of the concepts:
1) abstainers; 2) drinkers; 3) excessive drinkers; 4) alcoholics. That is, in the sense, in which these concepts are applied by Schmidt & De Lint (Schmidt & De Lint, 1970; De Lint & Schmidt, 1970, 1971).

Abstainers are considered those individuals of fifteen years and older, who drink alcoholic beverages less than once a year.

Drinkers are considered those individuals of fifteen years and older, who drink alcoholic beverages at least once a year.

Excessive drinkers are considered those individuals of fifteen years and older, whose mean daily consumption of absolute alcohol (100 vol.%) equals or surpasses 10 cl. ($= \pm$ 80 grams).

Alcoholics are considered those individuals of fifteen years or older, whose mean daily consumption of absolute alcohol (100 vol. %) equals or surpasses 15 cl. ($= \pm$ 120 grams).

The Ledermann equation is as follows:

$$t_{\wedge} = aZ_{\wedge} + \Theta$$

The ultimate unknown quantity measured by this formula, is the distribution of the percentages of drinkers according to their mean daily consumption of absolute alcohol (100 vol %) in cl or according to their mean annual consumption of absolute alcohol (100 vol %) in litres, this unknown quantity is P_{+s}.
P_{+s} is found via t_s, and the interrelation between these two magnitudes is explained further on

The Ledermann equation is composed of the following elements

Z_s = $^{10}\log x - {}^{10}\log D$

a = 2,302585 A

Θ = 3,43

x = mean daily volume of absolute alcohol (100 vol %) in cl, or mean annual volume of absolute alcohol (100 vol %) in litres, per drinker
x is the variable for which the percentage of drinkers drinking a mean daily/annual volume of x cl or litres of absolute alcohol, has to be determined, x varies from >0 to (theoretically) $+\infty$ and practically to a maximum of 365 litres of absolute alcohol annually, c q 100 cl ($= \pm$ 800 grams) daily

$$A = \frac{\Theta + \sqrt{\Theta^2 + 2Z'_M}}{-2Z'_M}$$

Z'_M = $^{e}\log m - {}^{e}\log D$

D = 365 litres per year

m = mean consumption of absolute alcohol (100 vol %) per drinker in a population, in litres per year

t_s = stochastic variable, pertaining to the standard normal distribution t_s can be found in tables of the standard normal distribution (Parzen, 1960, Diem & Lenther, 1970)

The Ledermann equation ($t_s = aZ_s + \Theta$) represents the relation between t_s and $^{10}\log x$ at a given value of m in a given population Hence for every value of x a value of t_s can be obtained With the aid of a table for the cumulative probabilities of the standard normal distribution (Parzen, 1960, Diem & Lenther, 1970) for every value of t_s the cumulative percentage pertaining to x can be found, Ledermann (1956) has called this cumulative percentage F_s

F_s = the proportion of drinkers - expressed in percentages - comprised in the interval of $-\infty$ to t_s, F_s implies a cumulative percentage, varying from 0% to approx 100%, for every value of t_s, and hence of x, F_s can be read in tables of the standard distribution

P_{+s} = the proportion of drinkers - expressed in percentages - who consume a daily mean of x cl of absolute alcohol (100 vol%)

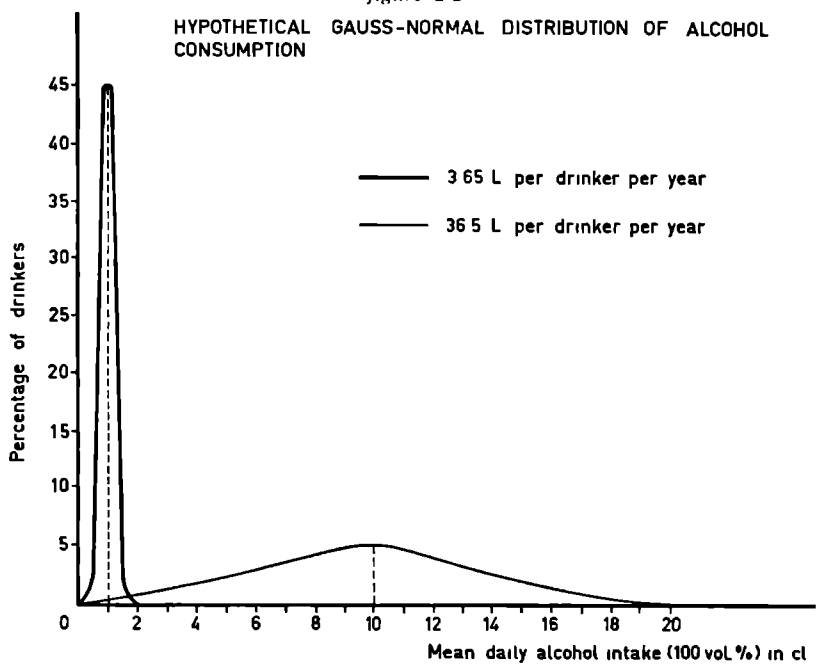
P_{+s} is found through F_s, by subtracting from every increasing value of F_s pertaining to a certain value of x the foregoing smaller value of F_s

As a contributory explanation the following comparison can be presented
In a population of recruits there exists an average body height In that population body height is normally distributed, and a certain percentage of recruits will have a body height of $\geq$ 2 00 metres In a different population of recruits with a greater average body height, the percentage of individuals with a body height of $\geq$ 2 00 metres will be greater than in the former population Ana-

logically the Ledermann equation implies that at a greater mean annual consumption of absolute alcohol in a population (m), the percentage of drinkers (P_{+}) who consume $\geq$ 150 ml of absolute alcohol as a daily mean ($x \geq 150$ ml) or $\geq$ 54 75 litres of absolute alcohol per year, will consequently be greater The difference, however, is that the Ledermann equation does not imply a Gauss-normal, but a lognormal distribution

The Ledermann equation implies, that the natural logarithms of alcohol consumption figures, and not alcohol consumption figures *per se*, are normally distributed This implies, that the distribution of the percentages of drinkers in a given population according to their mean daily consumption of absolute alcohol in cl, is represented by a lognormal curve This lognormal curve shows a peaking shape at a low mean annual alcohol consumption per drinker (e g 5 litres), and a flattened skewed shape at a high mean annual alcohol consumption per drinker (e g 25 litres) The reasoning followed by Ledermann (Ledermann, 1956, 1964) in the construction of his lognormal curve, is as follows With a (Gauss-)normal distribution of annual alcohol consumption the distribution of annual alcohol consumption would be predetermined in such a way, that in a population with a high mean annual alcohol consumption of e g 36 5 litres (= 100 ml or 10 cl or $\pm$ 80 grams daily), the maximally possible daily alcohol consumption would be 200 ml (= $\pm$ 160 grams), while at a low mean annual consumption of f i 3 65 litres (= 10 ml or 1 cl or $\pm$ 8 grams daily) this maximum would be 20 ml (= $\pm$ 16 grams) It is well known, however, that even in populations with low mean annual alcohol consumptions excessive drinkers and alcoholics do occur, i e drinkers with a mean daily alcohol consumption of $\geq$ 100 ml or $\geq$ 150 ml This implies, that the descending part of the curve is not compatible with the Gauss-normal distribution, but that it more or less asymptotically approaches the zero-line, cf *fig 2 2*

figure 2 2



Abstraction for fig 2.2

In a population with a low mean annual alcohol consumption (e.g. 3.65 litres) the average daily alcohol consumption is 1 cl (= ± 8 grams). If alcohol consumption would be (Gauss-)normally distributed as suggested in this figure, then there would be no drinkers in that population drinking in excess of 2 cl (= ± 16 grams) daily. Alcoholics consume ≥ 15 cl daily. However, even in populations with low mean annual alcohol consumption (e.g. 3.65 litres) alcoholics do occur. The descending part of the curve (for 3.65 litres) hence runs more to right than as suggested in this figure. Ledermann (Ledermann, 1956, 1964) therefore suggests a *log*normal distribution of annual alcohol consumption. In a population with a high mean annual alcohol consumption (e.g. 36.5 litres) the mean daily alcohol consumption is 10 cl (= ± 80 grams), and with a presumable Gauss-normal distribution the maximally possible daily alcohol consumption would be 20 cl (= ± 160 grams).

In populations with either low (3.65 litres) or high (36.5 litres) annual alcohol consumptions there are drinkers with a mean daily alcohol consumption of ± 25 cl (= ± 200 grams), namely alcoholics (Péquignot, 1958, Lelbach, 1966, Schmidt & Popham, 1968, Wilkinson et al., 1969). Thus for a high mean annual alcohol consumption the descending part of the curve also runs more to the right. Therefore for both high and low alcohol consumption Ledermann suggests a *log*normal distribution (Ledermann, 1956, 1964).

Ledermann maintains as a maximum a mean daily consumption of 1000 ml (± 800 grams) of absolute alcohol (100 vol %) or 365 litres (± 292 kg) of absolute alcohol per year, on the assumption that a higher consumption would have fatal consequences for the drinker (Ledermann, 1956, 1964).

Many aspects of human behavior have been adequately described by lognormal distributions. It has for instance been stated that the distribution of personal incomes is approximated by a lognormal distribution (Aitchison & Brown, 1966).

A mathematical exposition of the Ledermann equation is beyond the scope of this study. Besides, Hyland & Scott have compiled tables for the practical application of this equation (Hyland & Scott, 1969). With the aid of these tables the lognormal distribution and the lognormal curve can be constructed for a wide range of mean annual alcohol consumptions per capita of the drinking population (in litres per year). For the application of the Ledermann equation the following data must be know:

a) the total annual alcohol consumption in a given year in a given population,
b) the proportion of the "drinking population" (D.P.), this is found by subtracting the proportion of abstainers from the total number of the "drinking age population" (D.A.P.) i.e. the population of fifteen years or older,
c) the mean minimal daily alcohol consumption per alcoholic. This value may show regional variations but internationally the value of 15 cl (150 ml or 120 grams) is maintained as lower limit (De Lint & Schmidt, 1970, 1971, Schmidt & De Lint, 1970).

Lelbach mentioned a mean daily alcohol consumption of 23.3 cl (± 184 grams) in a representative sample of 526 alcoholics from a West-German clinic (Lelbach, 1966).

Wilkinson et al. mentioned a daily mean of 26.3 cl (± 208 grams) in a sample of 220 Australian alcoholics (Wilkinson et al., 1969).

Schmidt & Popham mentioned a daily mean of 25.4 cl (± 200 grams) among 100 alcoholics in a clinic in Toronto, Canada (Schmidt & Popham, 1968).

Pequignot mentioned a daily mean of 24.7 cl (± 195 grams) in a sample of patients with alcoholic liver cirrhosis in France (Pequignot, 1958).

The mean value in these four observations is 25 cl (250 ml or ± 200 grams), with 15 cl (150 ml or ± 120 grams) as lower limit.

The mean minimal daily alcohol consumption per *excessive drinker* is valued at 10 cl. (± 80 grams) (De Lint & Schmidt, 1970, 1971; Schmidt & De Lint, 1970).

2.1.3.2. Application of the Ledermann equation; Aruba, 1972-1973.

Data concerning total annual alcohol consumption were obtained by subtracting the total annual export of alcoholic beverages from the total annual import of alcoholic beverages. Aruba has no own production of alcoholic beverages on the basis of sugarcane cultivation like many Central American countries nor viticulture like France, Spain, Portugal, Italy and other Mediterranean countries nor beer-production from cereals like some European countries. All beverage alcohol in Aruba is imported, of which a negligible proportion is exported.

Data concerning annual import and export of alcoholic beverages were obtained through the cooperation of the Department of Social and Economic Affairs of the Netherlands Antilles (Jaarstatistiek van de in- en uitvoer per goederensoort van de Nederlandse Antillen; 1956-1976) and of the Department of Economic Development (DECO) in Aruba (DECO, 1970, 1970a, 1971).

Table 2.14. shows the computation of total annual alcohol consumption and of the annual alcohol consumption per capita of the drinking population (D.P.) for the year 1973, on the basis of the abovementioned data. In these computations the following data have been used:

a) According to the January 1972 Census (Eerste algemene volks- en woningtelling Nederlandse Antillen, 1972) in 1972 Aruba's population of fifteen years and older ("drinking age population", D.A.P.) amounted to 35,133, i.e. 57.3% of the total population.
b) According to the findings of this study (cf. *table 2.8.*) the percentage of abstainers in a D.A.P. sample amounts to 25.6%, hence the percentage of drinkers to 74.4%. Hence for 1972 the "drinking population" (D.P.) amounts to 74.4% x 35,133 = 26,139 or 42.6% of the total population.
c) According to recent data of the Department of Social and Economic Affairs (Statistisch Jaarboek Nederlandse Antillen, 1974) in 1973 Aruba's total population amounted to 61,717. Hence for 1973 the D.P. is estimated at 42.6% x 61,717 = 26,291.

d) In the computation of total annual alcohol consumption the following percentages for beer and wine (Merry, 1971) were employed: grape-must (12% - 13%); champagne and other sparkling wines (12%); wine (12% - 13%); vermouth (15%); cider and other fermented wines (7½%); beer (5%); stout (10% - 12%); malt-beer and other sparkling beer brewed from cereals (5%).

Table 2.14.

Computation of total annual alcohol consumption and of annual alcohol consumption per capita of the drinking population; Aruba, 1973.

Type of beverage.	Volume (litres)	Alcohol (100 vol.%) Volume (litres)
Import		
Champagne & other sparkling wines (12%)	8,723	1,047
Wine (12½ %)	127,672	15,959
Vermouth (15%)	19,136	2,870
Cider & other fermented wines (7½ %)	4,162	312
Beer (5%)	2,102,796	105,140
Amstel beer, Amstel Brewery Neth. Antilles (5%)	691,100	34,555
Stout (11%)	82,059	9,027
Malt beer & other sparkling beer from cereals (5%)	3,243	162
Cognac (50%)	48,111	24,056
Gin (50%)	13,268	6,636
Liqueur (50%)	30,845	15,423
Rhum (50%)	57,237	28,618
Whisky (50%)	267,920	133,960
Fermented alcohol, suitable for consumption (50%)	139,400	69,700
Dry gin (50%)	17,321	8,661
Vodka (50%)	12,633	6,317
Other distilled spirits (50%)	469	235
(Total)		462,676
Export		
Distilled spirits (50%) 5.000 kg.	6,329	— 3,165
		459,511
Per drinker: $\frac{459,511}{26,291}$ = 17.5 L		

e) For distilled spirits (cognac, gin, liqueur, rhum, whisky, dry gin, vodka, cucuy, etc) an alcohol percentage of 50% was employed, as used by the customs of the Netherlands Antilles (Jaarstatistiek van de in- en uitvoer per goederensoort van de Nederlandse Antillen, 1956-1976).

The total annual consumption of alcohol (100 vol.%) in 1973 amounted to 459,511 litres. Hence, in 1973 the mean annual alcohol consumption per drinker amounted to 459,511 / 26,291 = 17.5 litres; mean daily consumption amounted to 17,500 ml. / 365 = 48 ml.

In 1969 in France, the country with the highest mean annual alcohol consumption per capita of the drinking population, this mean amounted to 25.9 litres per drinker. The Netherlands had a mean annual consumption of 7.7 litres per drinker in 1967 (De Lint & Schmidt, 1970). With the aid of the Hyland & Scott tables (Hyland & Scott, 1969) the lognormal distribution of alcohol consumption in Aruba in 1973 is constructed; the figure 17.5 litres was rounded off at 18 litres.

The reason for maintaining the 1973 data instead of the 1972 data, is that it seemed reasonable to assume that in view of the high frequency of travelling abroad by Arubans and returning to the island generally without having to report beverage alcohol brought home from their trips, this "clandestine import" might produce an increase in the annual per capita consumption of alcohol.

Fig. 2.3. shows the lognormal distribution of alcohol consumption in Aruba in 1973.

Fig. 2.3. shows, that the percentage of drinkers who drink a daily mean of $\geqslant$ 15 cl. of absolute alcohol, amounts to *5.56%*, i.e. *1,462* among the 26,291 of the D.P. in 1973; according to the criteria indicated by De Lint & Schmidt this would be the percentage resp. the total number of alcoholics in this population (Schmidt & De Lint, 1970). The percentage of excessive drinkers among Aruba's D.P. in 1973 amounts to *11.51%*, or *3,026* among the 26,291.

Analogically, in *fig. 2.4.* the lognormal distributions of alcohol consumption for the Netherlands (1967) and for France (1969) have been constructed. The figures 7.7 L. and 25.9 L. were rounded off to 8 L. and 26 L. resp.

figure 2 3

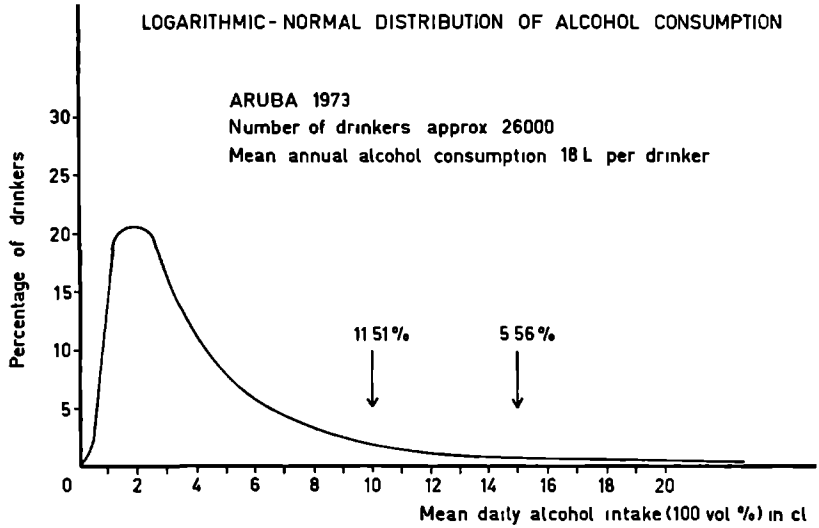


figure 2 4

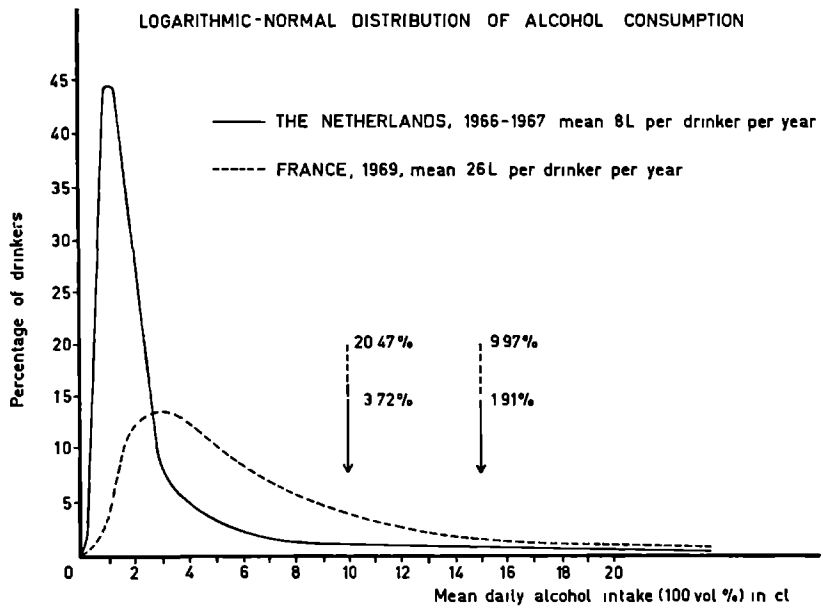


Fig. 2.4. shows, that in the Netherlands the percentage of drinkers who consume a daily mean of ⩾ 15 cl. of absolute alcohol (alcoholics) amounts to *1.91%*, while the percentage of excessive drinkers amounts to *3.72%*.

For France, these percentages are much higher; there the percentage of drinkers who consume a daily mean of ⩾ 15 cl. of absolute alcohol amounts to *9.97%*, and the percentage of excessive drinkers to *20.47%*.

2.1.3.3. Influence of tourism upon official alcohol consumption data.

With regard to total annual alcohol consumption it seems probable, that the official figures (Jaarstatistiek van de in- en uitvoer per goederensoort van de Nederlandse Antillen, 1956-1976) underestimate the real quantity. For the small population of Aruba *tourism* might have significant influence upon the official figures.

Table 2.15. shows a number of factors which may influence official alcohol consumption data.

Table 2.15.

Factors influencing total annual alcohol consumption without being detected by official registration of import and export of alcoholic beverages.

A) *Factors reducing annual alcohol consumption.*

1. The annual amount of alcohol bought, consumed or exported by tourists.
2. The annual amount of alcohol exported by Aruban travellers for instance as presents for people abroad.

B) *Factors increasing annual alcohol consumption.*

1. The annual amount of alcohol bought or consumed abroad or imported by Aruban travellers.
2. The annual amount of alcohol imported by tourists for instance as presents for people living in Aruba.

Nothing is exactly known about the factors mentioned in *table 2.15.* There are, however, official data concerning annual numbers of tourists, annual numbers of night stops by tourists, and annual numbers of arrivals (by aircraft and by ship) of local residents (Statistisch Jaarboek Nederlandse Antillen, 1974; Statistische Mededelin-

gen Nederlandse Antillen, 1970-1975; De ontwikkeling van het toerisme in de Nederlandse Antillen, 1974).

Table 2.16. shows these figures for 1964 and 1973.

Table 2.16.

Annual numbers of tourists, night stops by tourists, arrivals of local residents (by aircraft and by ship), and total population in 1964 and 1973.

Year	Numbers of tourists	Numbers of night stops by tourists	Numbers of arrivals by local residents	Total population
1964	20,247	133,903	35,636	59,858
1973	95,153	743,003	88,717	61,717

As shown in *table 2.16.*, the impact of tourism is enormous as compared with the total population. As may be seen in Chapter 3, there are reasons to hypothesize, that total annual alcohol consumption is larger than official alcohol consumption data, and that this is due to the factor B) 1. in *table 2.15.*

2.1.3.4. Trend-analysis of mean annual consumption, and annual prevalences of excessive drinkers and alcoholics.

Table 2.17. shows the course of the total population, the "drinking population" (D.P). for the years 1950 and 1956 through 1975, the prevalence rates of excessive drinkers (Ledermann equation), and the prevalence rates of alcoholics (Ledermann equation, Jellinek formula, population survey method). To this end data have been used from the Department of Social and Economic Affairs of the Netherlands Antilles (Statistisch Jaarboek Nederlandse Antillen, 1971; Statistische Mededelingen Nederlandse Antillen, 1970-1976). For the determination of the D.P. it was postulated that before and after 1972 the percentage of drinkers in the "drinking age population" (D.A.P.) amounted to 74.4% like in 1972, i.e. 42.6% of the total population. With the aid of the Ledermann equation the numbers of "excessive drinkers" and "alcoholics" have been calculated for each year. With the aid of the Jellinek formula the mean annual number of alcoholics has been calculated for the two five-year-periods 1961-1965 and 1966-1970.

Table 2.17

Total population, "drinking population" (D.P.), mean annual alcohol consumption, prevalence rates of "excessive drinkers", prevalence rates of alcoholics

Year	Total Population	D.P.	Mean annual alcohol consumption per drinker (litres/year)	Number of excessive drinkers (Ledermann)	Number of alcoholics (Ledermann)	Number of alcoholics (Jellinek) R = 3.5	Number of alcoholics (Jellinek) R = 5.3	Number of alcoholics (population survey method)
1950	±51,000	±21,700	11.4	1,226	611			
1956	56,050	23,877	14.1	1,894	926			
1957	57,213	24,373	13.6	1,933	946			
1958	58,486	24,915	15.8	2,402	1,166			
1959	58,868	25,078	14.9	2,198	1,071			
1960	58,743	25,025	15.3	2,193	1,069			
1961	57,347	24,429	12.7	1,742	855			
1962	58,506	24,924	13.9	1,977	967			
1963	59,315	25,268	12.2	1,612	793	807	1,219	
1964	59,858	25,500	13.0	1,818	893			
1965	59,415	25,311	11.8	1,615	795			
1966	59,323	25,272	15.6	2,436	1,183			
1967	59,020	25,143	14.8	2,202	1,074			
1968	59,231	25,232	14.0	2,001	979	1,311	1,982	
1969	59,813	25,480	14.8	2,234	1,088			
1970	60,734	25,873	16.5	2,730	1,322			
1971	60,811	25,905	16.3	2,498	1,212			
1972	61,293	26,139	16.1	2,520	1,223			2,530
1973	61,717	26,291	17.5	3,026	1,462			
1974	61,788	26,322	16.5	2,777	1,345			
1975	61,982	26,404	17.2	2,786	1,349			

The mean prevalence of alcoholism, calculated with the Jellinek formula for the decade 1961-1970, i.e. 1,603, appears to be 61% higher than the mean alcoholism prevalence, namely 995 calculated with the Ledermann equation. If one employs the mean value of R from 10 countries (U.S.A., Great Britain, Finland, Norway, Denmark, Sweden, Canada, France, Australia and Switzerland), namely 3.5, then the Jellinek formula produces the figure 1,059; in that case

there is almost complete concordance between the computations with both the Jellinek formula and the Ledermann equation.

With regard to the Jellinek formula, it has been stated, that this formula is never applicable to regions with *small* populations, and that results obtained with it, should be considered as only an approximation of the real figures (Argeriou, 1974; Efron & Keller, 1970; Keller, 1962; Lipscomb & Sulka, 1961).

With regard to the Ledermann model of alcohol consumption, it has been stated that this represents an attempt at describing and predicting alcohol consumption in *homogeneous* populations (Miller & Agnew, 1974).

The distribution of alcohol consumption in heterogeneous populations would *not* respond to the Ledermann equation. Skog found, that heterogeneous populations presented systematic deviations from the values indicated by the Ledermann model (Skog, 1973).

The Aruban population is a small, heterogeneous population, consisting of approx. 80% Arubans and approx. 20% others, originating from about 50 countries. Hence, the application of both the Jellinek formula and the Ledermann equation in *this* population meets with serious difficulties. From these specific circumstances in the Aruban population the discrepancy between the results obtained with the Jellinek formula and the Ledermann equation can be partially explained. It is remarkable too, that the Jellinek formula ($R = 5.3$) yields a number of 1,219 alcoholics for the five-year-period 1961-1965, and for the five-year-period 1966-1970 a number of 1,982. This would imply an increase of 63%, while the total population in that same period only shows an increase of approx. 1.5%, that is from a mean of 58,888 for the period 1961-1965 to a mean of 59,624 for the period 1966-1970. Probably this difference (63%) does not imply a real increase, but rather an improvement in the medical registration of mortality from (alcoholic) liver cirrhosis.

The annual figures calculated with the Ledermann equation present less extravagant fluctuations over the same periods (i.e. an increase of 31%, from a mean of 861 alcoholics for 1961-1965 to a mean of 1,129 for 1966-1970).

The annual fluctuations in mean annual alcohol consumption per drinker, and consequently, in the absolute numbers and percentages

of alcoholics, may rather reflect fluctuations in the conjuncture in the years 1956 through 1975. This conjuncture was and still is mainly determined by the oil refinery (Lago Oil and Transport Company), which in the nineteen-fifties started to lay off large numbers of employees, which consequently led to increasing unemployment rates. Only after the mid-sixties a modest change for the better started, among other things through the increasing of tourism.

There has been no explosive increase of mean annual per capita income, like in the Netherlands in the same period 1956-1975, but rather a standstill of mean per capita income.

The increase of mean alcohol consumption per capita of the D.A.P. in the Netherlands (Gips, 1975) from approx. 6 grams daily in 1950 to approx. 24 grams daily in 1974, may probably be ascribed, to a certain extent, to the increase in mean annual per capita income of the Dutch population through that period. During the same period in Aruba the mean daily alcohol consumption per drinker increased from 31.2 ml. (24.7 grams) to 48.0 ml. (37.8 grams).

figure 2.5.

TREND OF MEAN ANNUAL ALCOHOL CONSUMPTION ; ARUBA, 1950-1975

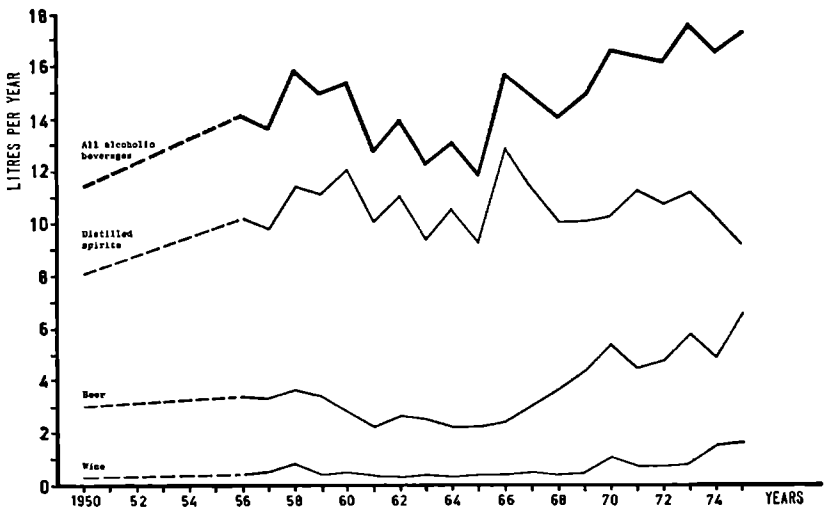


Fig. 2.5. shows mean annual consumption of all alcoholic beverages (in alcohol 100 vol.%), of beer (in alcohol 100 vol.%), of wine (in alcohol 100 vol.%) and of distilled spirits (in alcohol 100 vol.%) per capita of the D.P. for 1950 and for 1956 through 1975. It is evident, that the increase of mean alcohol consumption in Aruba from 1950 to 1975 amounts to 51%, i.e. 2% per year. In the Netherlands for example, this increase amounts to 306% for the same period, i.e. 12% per year (Compendium Gezondheidsstatistiek Nederland, 1974; Gips, 1975, 1976; Produktschap voor Gedestilleerde Dranken, 1971-1976).

Fig. 2.5. also shows that until the mid-sixties the ups and downs in total alcohol consumption were nearly entirely explicable by identical trends in consumption of *distilled spirits.* After 1965 there is a clear trend towards increasing alcohol consumption; this increase, however, is mainly due to increasing *beer* and *wine* consumption notwithstanding decreasing consumption of distilled spirits.

With a further increase of mean annual alcohol consumption in the Netherlands (Gips, 1975, 1976) it may be expected that both alcoholism prevalence and prevalence of deaths from alcoholic liver cirrhosis in the Netherlands will surpass the equivalent Aruban figures in the near future.

Table 2.18. shows mean annual alcohol consumption per capita of the D.A.P. in 1960 and 1970 for a number of countries (Produktschap voor Gedistilleerde Dranken, 1971, 1975, 1976; Solms, 1976; Wever & Gips, 1977). According to this list in 1960 Aruba belonged to the eleven countries with *major* alcohol problems, i.e. with mean annual alcohol consumptions above 10 litres per capita of the D.A.P. In 1970 Aruba appears among the fourteen countries with *major* alcohol problems. Between 1960 and 1970 the Netherlands for instance, have shifted from the group of countries with minor alcohol problems (i.e. below 5 litres) to those with *moderate* alcohol problems (i.e. between 5 and 10 litres). In 1975 these figures were: 12.8 litres for Aruba, and 11.6 litres for the Netherlands (Wever & Gips, 1977).

Table 2.18

Mean annual alcohol consumption (in litres of alcohol 100 vol %) per capita of the D.A.P. in 1960 and 1970 for a number of countries

1960		*1970*	
France	27.3	France	24.0
Italy	19.1	Italy	20.7
Portugal	15.3	Spain	16.9
Luxemburg	13.8	Luxemburg	16.2
Switzerland	12.6	Federal Republic of Germany	16.0
Spain	11.9	Portugal	15.7
Belgium	11.7	USSR	14.6
Aruba	11.4	Switzerland	14.5
Austria	10.9	Austria	13.3
USSR	10.4	Belgium	13.2
Federal Republic of Germany	10.2	Hungary	13.0
Hungary	9.2	Aruba	12.3
U.S.A.	7.8	German Democratic Republic	10.5
German Democratic Republic	7.3	Yugoslavia	10.4
Yugoslavia	6.8	U.S.A.	9.7
Great Britain	6.8	Denmark	9.7
Poland	6.2	Great Britain	8.3
Denmark	6.1	Sweden	7.9
Sweden	5.9	The Netherlands	7.8
Ireland	4.9	Poland	7.5
Finland	3.9	Ireland	7.3
The Netherlands	3.7	Finland	6.3
Norway	3.6	Norway	4.4

2.2. COMPARISON BETWEEN THE RESULTS OBTAINED WITH THE POPULATION SURVEY METHOD, THE JELLINEK FORMULA AND THE LEDERMANN EQUATION

Since underestimation of the prevalence of alcoholism for the purpose of planning of therapeutic and preventive measures in the near future is less favorable than overestimation, the comparison will be drawn between the maximum estimates obtained with the three methods employed. As can be concluded from *table 2.17.*, the mean number of alcoholics estimated with the Ledermann equation for 1961 through 1965 is 861, while this figure for 1966 through

1970 is 1,129. This implies an increase of *31%*, or *6.2%* per annum. This annual increase of *6.2%* is also presumed to be applicable for the Jellinek formula, since the annual increase (12.5%, or 63% for five years) found with this formula was rather considered to reflect improved medical registration of (alcoholic) cirrhosis mortality.

For the period 1961-1965 the mean number of alcoholics estimated with the Jellinek formula ranges from 807 (at R = 3.5) to 1,219 (at R = 5.3).

For the period 1966-1970 this figure ranges from 1,311 (at R = 3.5) to 1,982 (at R = 5.3).

For the period 1971-1975 this figure is *extrapolated:* 1,700 (at R = 3.5) or 2,600 (at R = 5.3), since there were no data available concerning liver cirrhosis mortality for 1971 through 1975.

Table 2.19. shows the comparison between the three methods.

Table 2.19.

Comparison between the results obtained with the population survey method, the Jellinek formula, and the Ledermann equation.

Method	Number of alcoholics	Prevalence of alcoholism per 10,000 of the D.A.P.	Prevalence of alcoholism per 10,000 of the D.P.	Prevalence of alcoholism per 10,000 of the total population
Population survey method, 1972	2,530 (range 1,860-3,200)	720	968	413
Jellinek formula, 1971-1975	1,700 (R=3.5)	484	651	277
(extrapolated)	2,600 (R=5.3)	740	995	424
Ledermann equation, 1971-1975	1,318	374	503	214

Table 2.19. shows that for 1972 and for 1971-1975 the number of alcoholics in Aruba can be estimated at approx. 1,300-3,200. There are significant discrepancies between the results obtained with the three methods.

With regard to the *Jellinek formula* it has been stated that this method is less reliable in *small* populations (Argeriou, 1974; Efron & Keller, 1970; Keller, 1962; Lipscomb & Sulka, 1961). With regard to the *Ledermann equation* it has been stated that this method is less reliable in *heterogeneous* populations (Miller & Agnew, 1974; Skog, 1973).

The *Ledermann equation* is also less reliable in "detecting" *inactive alcoholism:*
individuals who meet with the criteria for the diagnosis of alcoholism as described in this chapter, but who drink less than 15 cl. of alcohol (100 vol.%) daily.

Moreover it is debatable, whether this lower limit ($\geqslant$ 15 cl. of absolute alcohol daily) is adequate. Schmidt & Popham, for instance, mention a lower limit of *14 cl.* (Schmidt & Popham, 1968), Lereboullet *12.5 cl.* among 2,749 respondents (Lereboullet, 1964), and Lundquist even *8.5 cl.* (Lundquist, 1972) among 200 male respondents. Hence the question remains whether this lower limit should be set at a lower level, for instance 10 cl. ("excessive drinking", like previously mentioned) or whether this lower limit varies from one population to another.

With regard to the *population survey method* it can be suggested that it would imply *over*estimation because its diagnostic criteria would possibly include too broad a definition of alcoholism. This has been carefully avoided by using a *narrower* definition - as compared to recent publications - cf. table 2.3. (American Psychiatric Association, 1968; Criteria Committee, 1972; Van Epen, 1974; Jellinek, 1960; Jones et al., 1970). With regard to the *population survey method* as applied in this study, not only active but also *inactive alcoholism* (Knupfer, 1967) is detected: alcoholics who in a certain period, for instance a year, drink no or less than 15 cl. of alcohol (100 vol.%) daily, because of such events like protracted detention or illness or because of (temporary) adequate treatment. This is due to the fact that in most variables used to detect alcoholism (or its precursors stages) the *time factor* was not evaluated, i.e. little or no information was gathered concerning the *period* in which those problems were present for the respondent.

2.3. CONCLUSIONS

1) With the population survey method the number of alcoholics in Aruba in 1972 can be estimated at 2,530, with as 95%-confidence interval 1,860-3,200.

2) With the three methods employed the number of alcoholics in Aruba can be estimated at approx. 1,300-3,200, i.e. 5.0%-12.2% of the D.P., 3.7%-9.1% of the D.A.P., and 2.1%-5.2% of the total population.

3) In this relatively small population (61,293 in 1972) there is *no* optimal concordance between the estimations of alcoholism prevalence with the population survey method, the Jellinek formula and the Ledermann equation. This is due to:
   a) The small number of the total population (Jellinek formula);
   b) Possible underreporting of (alcoholic) liver cirrhosis mortality (Jellinek formula);
   c) The heterogeneity of the population (Ledermann equation);
   d. The significant influence of tourism upon official alcohol consumption data possibly leading to underestimation of total alcohol consumption (Ledermann equation);
   e) The questionableness of the level of 15 cl. of absolute alcohol daily as lower limit for alcohol consumption by alcoholics (Ledermann equation); these internationally accepted lower limit levels (15 cl., 10 cl.) for the Ledermann equation have been chosen rather arbitrarily, thereby debilitating the importance of the formula;
   f. The fact that inactive alcoholism is detected by the population survey method but not by the Ledermann equation.

4) *Formulas* employed for estimating prevalences generally imply a rather global approach, hence leading to rougher estimates as compared to the population survey method, which - especially in small populations - is based on exact measurements of these prevalences.

DESCRIPTIVE EPIDEMIOLOGY: FUNCTIONAL CHARACTERISTICS OF ABSTINENCE, DRINKING AND EXCESSIVE DRINKING

3.1. INTRODUCTION

As stated in Chapter 1, descriptive epidemiology describes prevalences and incidences of disease, death or health and of characteristics associated with these three phenomena. In Chapter 2 solutions were proposed for the question "Which disease(s) or phenomena?" (determination of prevalences of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking, social drinking, and abstinence).

In Chapter 3 solutions will be proposed for the following questions:

1) Amount of drinking:
   a. Frequencies of alcohol consumption and intoxication;
   b. Daily/annual volumes of alcohol consumption;
   c. Monthly expenses on beverage alcohol;
   d. Beverage preference;
   e. Duration of drinking bouts;
   f. Weekend excesses of drinking and smoking;
   g. Cultural crisis of excessive drinking;
2) Phaseology of drinking;
3) Tolerance;
4) Age disribution;
5) Public opinions;
   a. Opinions concerning the alcoholic;
   b. Opinions concerning AA work.

3.2. AMOUNT OF DRINKING

3.2.1. Frequencies of alcohol consumption and intoxication.

For the questions "How often do you drink beverage alcohol?"

(variable no. 36) and "How often are you alcohol intoxicated?" (variable no. 37) the possible responses, estimated annual frequencies and assigned scores, are shown in *table 3.1.*

Table 3.1.

Estimated annual frequencies and assigned scores for frequency of alcohol consumption (variable no. 36) and frequency of alcohol intoxication (variable no. 37).

Answers	Estimated annual frequency	Score
Never	0	0
Once yearly or less	1	1
At parties only	14	3
Every two months	6	2
Once weekly	52	4
Every weekend	156	5
2-3 times weekly	156	5
Daily	365	6
Not applicable	0	0

The presumed annual frequency for the response "at parties only" was estimated at 14, on the basis of: 2 Christmas days, 1 New Year's Day, 3 Carnival days, 3 Easter Days, Queen's Birthday (April 30), Ascension Day, 3 Pentecost days (cf. variable no. M). The presumed annual frequency for the response "every weekend" was estimated at 156, on the basis of 3 weekend days)Friday through Sunday) multiplied by 52, the number of weeks in one year. The presumed annual frequency for the response "2-3 times weekly" was also estimaed at 156, on the basis of a maximum of 3 weekdays multiplied by 52, the number of weeks in one year.

In *table 3.1.* scores were assigned according to the sequence of estimated magnitudes of annual frequencies.

Table 3.2. shows mean annual frequencies of alcohol consumption and alcohol intoxication in the 6 diagnostic categories in survey A.

Fig. 3.1. depicts *table 3.2.* data graphically.

Table 3.2.

Mean annual frequency of alcohol consumption (variable no. 36) and mean annual frequency of alcohol intoxication (variable no. 37) in the six diagnostic categories in survey A (N = 708)

Diagnostic category	Number of respondents (N)	Mean annual frequency of alcohol consumption		Mean annual frequency of alcohol intoxication	
		In days per year	In scores	In days per year	In scores
Abstinence	181	0	0	0	0
Social Drinking	371	43	3.8	1	1.0
Problem Drinking	83	64	4.1	13	2.9
Prealcoholism	6	84	4.3	8	2.3
Gamma-prealcoholism	16	82	4.3	13	2.9
Alcoholism	51	113	4.6	36	3.6
(Total)	(708)	41	3.7	5	1.8

figure 3.1.

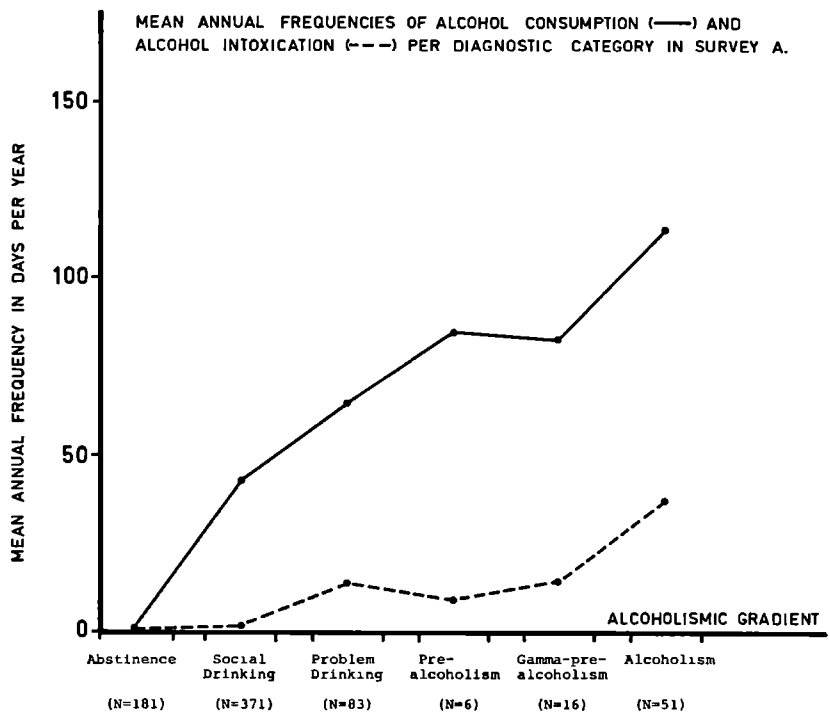


Table 3.3. shows mean annual frequencies of alcohol consumption and alcohol intoxication in survey B & C.

Table 3.3

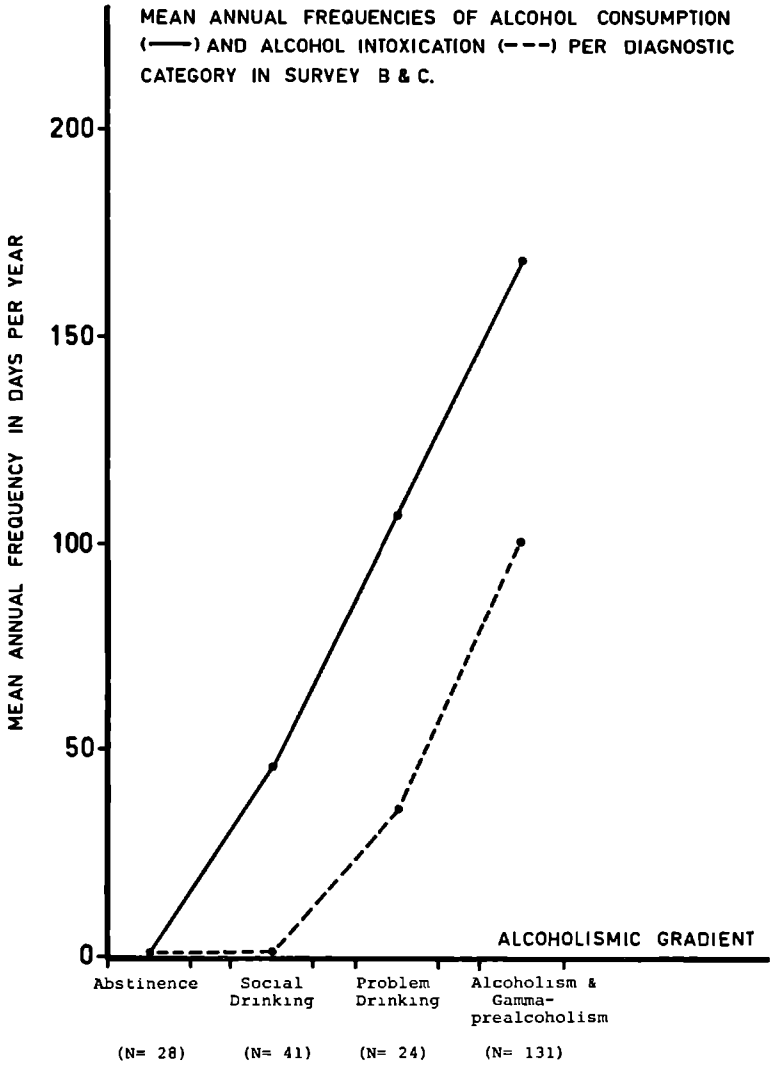
Mean annual frequency of alcohol consumption (variable no. 36) and mean annual frequency of alcohol intoxication (variable no. 37) in survey B & C (N = 224)

Diagnostic category	Number of respondents (N)	Mean annual frequency of alcohol consumption		Mean annual frequency of alcohol intoxication	
		In days per year	In scores	In days per year	In scores
Abstinence	28	0	0	0	0
Social Drinking	41	46	3.8	1	1.0
Problem Drinking	24	107	4.5	35	3.6
Alcoholism (& Gamma-prealcoholism)	131	169	5.1	99	4.5

Fig. 3.2. depicts *table 3.3.* data graphically.

Both *table 3.2.* and *table 3.3.* show, there is a gradual increase in frequencies of alcohol consumption and of alcohol intoxication on the alcoholismic gradient. As mentioned in Chapter 2, both these variables (frequency of alcohol consumption; frequency of alcohol intoxication) were used in the construction of the factors "alcoholism" and "problem drinking". Hence it is not surprising to find the correlations as shown in *fig. 3.1.* and *fig. 3.2.* It implies that *excessive drinking (heavy drinking)* increases gradually on the alcoholismic gradient, a definitional requisite proposed by Jellinek (1960).

figure 3.2.



3.2.2. Daily/annual volumes of alcohol consumption.

Table 3.4. shows mean daily and annual volumes of alcohol consumption (variable no. M) per diagnostic category.

Table 3.4.

Mean daily (in ml.) and annual (in litres) volumes of alcohol consumption (variable no M) per diagnostic category

Diagnostic category	N	Mean daily alcohol consumption in ml 100 vol % alcohol	Range (ml)	Mean annual alcohol consumption in litres 100 vol % alcohol	Range (litres)
Social Drinking	33	19	1- 139	7	0 4-50 7
Problem Drinking	20	48	1- 233	17 4	0 4-85
Gamma-prealcoholism	4	100	63- 180	36 3	23 -65.7
Alcoholism	116	278	30-1843	101 4	11 -673

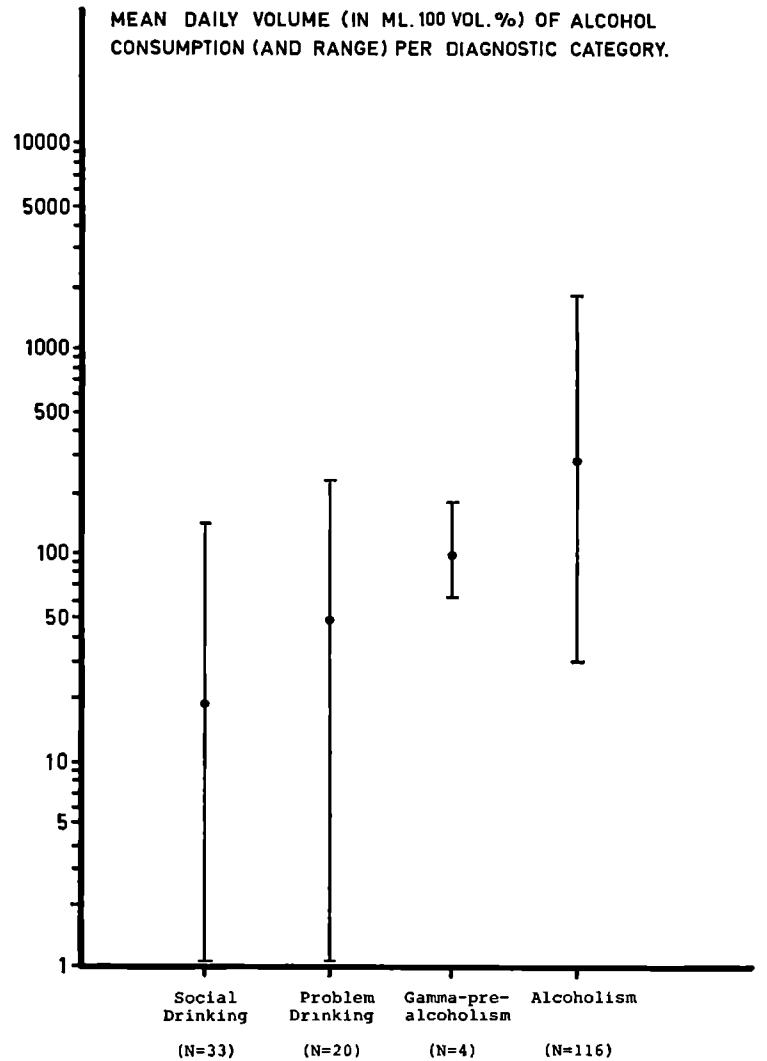
Though the *mean* daily and annual volumes of alcohol consumption are definitely different for the four diagnostic categories and in agreement with the alcoholismic gradient, as shown in *table 3.4.*, the *ranges* are equally impressive. It can be seen for instance, that at least among this study's group of 116 *treated* alcoholics, the lower limit is *30 ml.* of alcohol 100 vol.% daily and *not* 150 ml. as postulated in other studies (De Lint & Schmidt, 1970, 1971; Schmidt & De Lint, 1970). Of course this may be explained by assuming that, when once having been treated, many alcoholics are no longer completely able or willing to accurately estimate their daily/annual alcohol consumption prior to treatment, perhaps because their episode of excessive drinking is too far away in their memories. In addition, this may be explained by the assumption, that alcoholics seen at alcoholism clinics usually are no longer in the group of steady (excessive) drinkers i.e. they no longer belong to the "active alcoholics" (De Lint, 1975; Knupfer, 1967). Hence their daily volume of alcohol intake may vary considerably.

In an attempt to overcome this difficulty, a second selection was made, and only alcoholics who: 1) admitted having been drunk as often or more often as compared to the period before they were treated and 2) admitted having drunk as much or more after

treatment as compared to the pretreatment period, were evaluated (variables no. 173 and 166 resp.).

In this manner twelve alcoholics remained, with a mean daily consumption of 254 ml., an upper limit of 490 ml. and a lower limit

figure 3.3.



of 69 ml. Still the 150 ml. value (Schmidt & De Lint, 1970) was not reached, but this value of 69 ml.. approaches the one (85 ml.) described in another study (Lundquist, 1972). Thus it seems that the 150 ml. as lower limit (De Lint & Schmidt, 1970, 1971; Schmidt & De Lint, 1970) is questionable. The question arises whether this limit has to be *lowered* to a lower value, or whether this lower limit would be a variable in different cultures and populations.

The validity of self-reported consumption estimates of the type used in this study, has been questioned (De Lint, 1975), and frequency distributions of alcohol buying by individuals as officially reported by retail dealers were considered more accurate to estimate volume of alcohol consumption in Ontario (De Lint & Schmidt, 1968).

Fig 3.3. shows mean daily volumes of alcohol consumption per diagnostic category.

An extrapolation of the mean annual alcohol consumptions per diagnostic category to the real numbers of these categories in Aruba's D.P. gives as a result that the per drinker annual alcohol consumption in 1972 was 19.0 L. - which is higher than the 16.1 L. as derived from official figures. This problem was dealt with in Chapter 2.

Table 3.5.

Familial monthly expenses on beverage alcohol (variable no. 19) in percent.

Diagnostic category	N	Familial monthly expenses (NAfl.) on beverage alcohol in percent			
		NAfl. 0 score 1	NAfl. 1-50 (median & mode) score 2	NAfl. 50-100 score 3	⩾ NAfl. 100 score 4
Abstinence	25*	68	32	—	—
Social Drinking	41	42	56	2	—
Problem Drinking	24	21	58	21	—
Alcoholism (& Gamma-prealcoholism)	128*	12	45	23	20

$\chi^2 = 37.95$, df = 3, $p < 0.001$ — After dichotomization in *above* or *below* median scores.

* "Not applicable" respondents were omitted (three abstainers, three alcoholics).

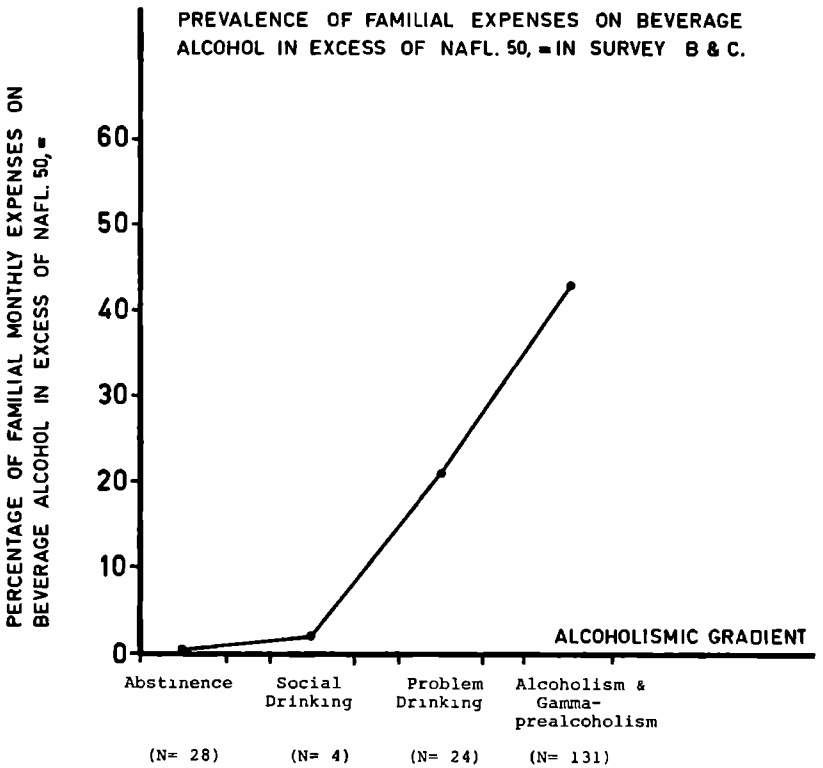
3.2.3. Monthly expenses on beverage alcohol.

Table 3.5. shows the distribution of familial monthly expenses on beverage alcohol in 4 diagnostic categories in survey B & C.

Fig. 3.4. depicts *table 3.5.* data graphically.

In *fig. 3.4.* the percentages of above median scores is shown per diagnostic category; median is: score 2, i.e. NAfl. 1-50.

figure 3.4.



The results shown in *table 3.5.* and *fig. 3.4.* indicate, that there is a gradual increase in monthly expenses on beverage alcohol on the alcoholismic gradient. This variable (familial monthly expenses on beverage alcohol), however, measures *amount of drinking* in the *family* of a respondent *including* the respondent. This explains why

even among abstainers 32% recognize to have monthly expenses on beverage alcohol: this expenditure is presumably ascribable to the nonabstaining relatives of the abstaining respondents.

It is evident ,that this variable *indirectly* measures amount of drinking, whilst "frequency of alcohol consumption", "frequency of alcohol intoxication" and "daily/annual volume of alcohol consumption" imply direct measurements. Still, this indirect measurement was usefully employed by Schmidt & De Lint to determine mean annual alcohol consumption per drinker in the drinking population of Ontario (Canada) and was found more reliable than direct measurements (De Lint, 1968; De Lint & Schmidt, 1968; Schmidt & De Lint, 1970).

The results shown in *table 3.5.* and *fig. 3.4.* also imply, that *excessive drinking* (= heavy drinking, according to Cahalan et al.) (Cahalan & Cisin, 1968; Cahalan et al., 1969) increase gradually on the alcoholismic gradient, a definitional requisite proposed by Jellinek (1960).

3.2.4. Beverage preference.

Table 3.. shows the distribution of beverage preference in the six diagnostic categories in survey A.

Table 3.6.

Beverage preference (variable no. 30) in percent, in survey A (N = 708).

Diagnostic category	N	Beverage preference			
		Soft drinks (Coca Cola, Juices, etc.)	Beer	Wines, sherry or vermouth	Hard liquor (Rhum; brandy; cucuy; whisky; liqueur; gin, etc.)
		Score 0	Score 1	Score 2	Score 3
Abstinence	181	100	—	—	—
Social Drinking	371	28	36	13	23
Problem Drinking	82*	32	40	8	20
Prealcoholism, Gamma-prealcoholism & alcoholism	73	21	38	4	37

* "Not applicable" responses omitted (one problem drinker).

Table 3.7. shows the distribution of beverage preference in the diagnostic categories in survey B & C.

Table 3.7

Beverage preference (variable no 30) in percent, in survey B & C (N = 224).

Diagnostic category	N	Beverage preference			
		Soft drinks Score 0	Beer Score 1	Wines Score 2	Hard liquor Score 3
Abstinence	28	100	—	—	—
Social Drinking	41	24	39	2	34
Problem Drinking	24	12	63	4	21
Alcoholism (& Gamma-prealcoholism)	130*	2	25	5	69

* "Not applicable" responses omitted (one alcoholic).

The results as shown in *table 3.6.* and *table 3.7.* indicate that:

a) Among *social drinkers* and *problem drinkers* the preferred alcoholic beverage is *beer;*
b) Among *alcoholics* and *gamma-prealcoholics (table 3.7.)* the preferred alcoholic beverage is *hard liquor* (rhum, brandy, cucuy, whisky, liqueur, gin, etc.).

Hence it is evident, that there is a gradual shift in preference of beverage alcohol from beverages with *low alcohol vol.%* to beverages with *high alcohol vol.%* on the alcoholismic gradient.

These findings contrast those of Cahalan et al., who found heavy drinkers more likely to be heavy drinkers of beer than of spirits or wine (Cahalan et al., 1969). These findings also contrast the statements by Schmidt & De Lint, that populations of alcoholics do *not* differ significantly from the drinking population at large as regards beverage preference (De Lint & Schmidt, 1971, 1971a, 1971b). In survey B & C (N = 224) the various types of hard liquor are preferred differently according to the various diagnostic categories; this is visualized in *table 3.8.*

Table 3.8

Beverage preference for various types of hard liquor (variable no 30) in percent, in survey B & C (N = 224)

Diagnostic category	N	Beverage preference					
		Rhum	Brandy	Cucuy	Whisky	Liqueur	Gin, etc
Social Drinking	41	2	—	—	25	—	7
Problem Drinking	24	8	—	—	8	—	4
Alcoholism (& Gamma-prealcoholism)	131	46	5	2	12	1	3

The results in table 3.8. indicate:

a) *Whisky* is the preferred type of hard liquor among social drinkers,
b) *Rhum* is the preferred type of hard liquor among alcoholics and gamma-prealcoholics;
c) Among problem drinkers whisky and rhum are equally preferred;
d) On the alcoholismic gradient there is a shift from more expensive hard liquor (whisky) among social drinkers to cheaper types of hard liquor (rhum, brandy) among alcoholics and gamma-prealcoholics.

Abstainers were omitted from the comparison in *table 3.8.*, since they do not drink alcoholic beverages.

Hence it is evident from the results in tables 3.6., 3.7. and 3.8., that with increasing alcoholismic gradient, drinkers tend to choose drinks which imply higher concentrations of alcohol (in vol.%) and to choose cheaper hard liquor, in order to get more alcohol for less money; these findings are concordant with the increase in amount of drinking on the alcoholismic gradient, as described in the preceding sections concerning frequencies of alcohol consumption and intoxication, volume of alcohol consumption, and monthly expenses on beverage alcohol.

3.2.5. Mean duration of drinking bouts.

Table 3.9. shows the distribution of mean duration of drinking bouts.

Table 3.9.

Mean duration of drinking bouts (variable no. 61) in percent, in survey B & C (N = 224).

Diagnostic category	N	Mean duration of drinking bouts				
		1-2 days	3-7 days	3-7 weeks	3-7 mths.	⩾ 1 year
Social Drinking	2*	100	—	—	—	—
Problem Drinking	8*	88	12	—	—	—
Alcoholism (& Gamma-prealcoholism)	121*	39	36	10	1	14

* "Not applicable" responses were omitted (39 social drinkers, 16 problem drinkers, 10 alcoholics, and all 28 abstainers)

The results in *table 3.9.* indicate that with increasing alcoholismic gradient, drinking bouts (periods of prolonged excessive drinking with drunkenness) tend to grow longer; this finding is concordant with the increase in amount of drinking on the alcoholismic gradient, as described in the preceding sections concerning frequencies of alcohol consumption and intoxication, volume of alcohol consumption, monthly expenses on beverage alcohol, and beverage preference.

3.2.6. Weekend excesses of drinking and smoking.

3.2.6.1. Weekend excesses of drinking.

Table 3.10. shows the distribution of weekly rhythm of excessive drinking.

Table 3.10.

Weekly rhythm of excessive drinking (variable no. 34) in percent, in survey B & C (N = 224)

Diagnostic category	N	Weekend (Friday, Saturday, Sunday)	Week days (Monday through Thursday)	Not applicable
Abstinence	28	11	4	86
Social Drinking	41	29	—	71
Problem Drinking	24	58	—	42
Alcoholism (& Gamma-prealcoholism)	131	73	5	22

The results shown in *table 3.10.* indicate that weekend drinking appears to be a prominent feature of both problem drinking and alcoholism, but *not* of social drinking or abstinence. Moreover the prevalence of weekend drinking *increases* on the alcoholismic gradient. This is in concordance with other studies (Gadourek, 1963) and with previously reported weekend excesses of problems associated with drinking by the Police Department of Aruba (Janssen, 1971; Oldenboom, 1971).

Table 3.10. also shows that *15%* of *abstainers* admit drinking on weekends or week days; this may be explained by assuming that this reflects former drinking habits by abstainers who once were drinkers, or possibly by the respondent's misunderstanding of variable no. 34 in the sense that also drinking of non-alcoholic beverage was meant.

Table 3.11. shows the distribution of *weekly rhythm of excessive drinking* in survey A sample.

Table 3.11.

Weekly rhythm of excessive drinking (variable no. 34) in percent, in survey A (N = 708).

Diagnostic category	N	Weekend	Week days	Not applicable
Abstinence	181	7	1	92
Social Drinking	371	35	1	64
Problem Drinking	83	52	1	47
Prealcoholism, Gamma-prealcoholism & Alcoholism	73	58	1	41

In survey A (cf. table 3.11.) the same trends are discernible as in survey B & C (cf. table 3.10.).

3.2.6.2. Weekend excesses of smoking.

Table 3.12. shows the distribution of *weekly rhythm of excessive smoking.*

Table 3 12

Weekly rhythm of excessive smoking (variable no 27) in percent, in survey B & C (N = 224)

Diagnostic category	N	Weekend	Week days	Not applicable
Abstinence	28	—	—	100
Social Drinking	41	10	—	90
Problem Drinking	24	13	4	83
Alcoholism (& Gamma-prealcoholism)	131	38	2	60

The results in *table 3.12* indicate, that weekend smoking is *not* characteristic for any of the diagnostic categories, (since its prevalence is always $< 50\%$), though the prevalence of weekend smoking increases on the alcoholismic gradient.

Table 3.13 shows the distribution of *weekly rhythm of excessive smoking* in survey A sample.

Table 3 13

Weekly rhythm of excessive smoking (variable no 27) in percent, in survey A (N = 708)

Diagnostic category	N	Weekend	Week days	Not applicable
Abstinence	181	4	—	96
Social Drinking	371	12	1	88
Problem Drinking	83	16	2	82
Prealcoholism, Gamma-prealcoholism & Alcoholism	73	31	—	69

The results in *table 3.13* indicate, that in survey A the same trend is discernible as in survey B & C.

3.2 7. Cultural crisis of alcohol consumption.

Table 3 14. shows the mean factor scores for this factor.

Table 3.14.

Mean factor scores (± S.E.M.) of factor "cultural crisis"; Pearson correlation coefficient (R); level of significance (p).

Diagnostic category	N	Mean factor score	S.E.M.
Abstinence	28	402	0
Social Drinking	41	490	15
Problem Drinking	24	511	21
Alcoholism (& Gamma-prealcoholism)	131	522	9

R = 0.3193
p = 0.001

The factor *cultural crisis of alcohol consumption* (Horwitz et al., 1967) is composed of variables no. 53, 54, 55, 56 & 57. It reflects excessive drinking on national holidays, religious holidays, family parties, paydays and during carnival festivities. It implies, that apart from weekend excesses (as shown previously), there are also other culturally determined occasions for excessive drinking. The findings suggest, that increasing alcoholismic gradient implies increasing drinking excesses on such culturally determined occasions.

Table 3.15. shows the variables included in this factor, and factor loadings per variable.

Table 3.15.

Factor analysis of the factor "cultural crisis of alcohol consumption".

Variable No.	Content of variable	Factor loading
53	Excessive alcohol consumption on national holidays like the Queen's birthday	0.8507
54	Idem on religious holidays like Christmas, Easter, Pentecost	0.7328
55	Idem on family parties	0.8271
56	Idem on paydays	0.5198
57	Idem during Carnival	0.8623

3.3. PHASEOLOGY OF DRINKING

3.3.1. Age at first drink.

Table 3.16. shows the distribution of *age at first drink.*

Table 3 16

Age at first drink (variable no 38) in percent

Diagnostic category	N	Age at first drink			
		10-14	15-19	20-24	⩾ 25
Abstinence	1*	100	—	—	—
Social Drinking	20*	5	50	35	10
Problem Drinking	23*	17	44	39	—
Alcoholism (& gamma-prealcoholism)	130*	4	60	25	12

* "Not applicable" responses were omitted (27 abstainers, 21 social drinkers, one problem drinker, one alcoholic)

Table 3.16. shows that *modal* and *median ages at first drink* differ very little among the diagnostic categories: *modal* age at first drink is 15-19 years, and *median* age between (15-19 years) and (20-24 years).

Among the total group in survey B & C, only 5% admit having had their first drink before age 15, this percentage may be extrapolated to the total D.A.P., since after adjusting the proportions of the diagnostic categories to those found in survey A (cf. Chapter 2) the same percentage (5%) emerges.

3.3.2. Age at first intoxication.

Table 3 17. shows the distribution of *age at first intoxication.*

Table 3 17

Age at first intoxication (variable no 39) in percent

Diagnostic category	N	10-14	15-19	20-24	25-29	30-34	⩾ 35
Social Drinking	9*	—	11	78	11	—	—
Problem Drinking	20*	5	30	45	15	5	—
Alcoholism (& Gamma-prealcoholism)	128*	3	48	31	9	5	5

* "Not applicable" responses were omitted (32 social drinkers, 4 problem drinkers, 3 alcoholics, and all 28 abstainers)

Table 3.17. shows that *median ages at first intoxication* differ somewhat between the diagnostic categories:

a) For alcoholics and gamma-prealcoholics *median* age at first in-

toxication is 15-19 years, while for problem drinkers and social drinkers this is 20-24 years; hence, alcoholics (and gamma-prealcoholics) appear to have experienced intoxication at a younger age;

b) For the total group in survey B & C *median* age at first intoxication is between (15-19 years) and (20-24 years). *11%* of social drinkers, *35%* of problem drinkers and *51%* of alcoholics and gamma-prealcoholics experienced their first intoxication at an age below this median.

3.3.3. Duration of social drinking before inception of problem drinking.

Table 3.18. shows the distribution of *duration of social drinking before inception of problem drinking.*

Table 3.18.

Duration of social drinking before inception of problem drinking (variable no. 40) in percent.

Diagnostic category	N	Duration of social drinking (in years)						
		0-1	1-3	3-5	5-9	10-14	15-19	⩾ 20
Social Drinking	1*	100	—	—	—	—	—	—
Problem Drinking	8*	37	13	13	13	13	—	13
Alcoholism (& Gamma-prealcoholism)	125*	11	23	11	18	13	12	11

* "Not applicable" responses were omitted (40 social drinkers, 16 problem drinkers, 6 alcoholics, and all 28 abstainers).

The following findings are shown in *table 3.18:*

a) For problem drinkers *median* duration of social drinking before inception of problem drinking is between 1-3 years and 3-5 years.

b) For alcoholics and gamma-prealcoholics *median* duration of social drinking before inception of problem drinking is between 5-9 years and 10-14 years. This difference between (gamma-pre-) alcoholics and problem drinkers is contrary to exceptation: one would have expected *shorter* (or equal) duration of social drinking among alcoholics and gamma-prealcoholics, since problem drinking generally starts *before* alcohol addiction. An alternative explanation: perhaps alcohol addicts experience the onset of *addiction* as inception of problem drinking.

c) Among social drinkers one respondent admitted the onset of problem drinking; this may imply a case of once having been a problem drinker and afterwards returning to social drinking.

3.3.4. Psychological dependence.

3.3.4.1. Age at onset of psychological dependence.

Table 3.19. shows the distribution of *age at onset of psychological dependence.*

Table 3.19.

Age at onset of psychological dependence (variable no. 59) in percent.

Diagnostic category	N	Age at onset of psychological dependence							
		15-19	20-24	25-29	30-34	35-39	40-44	45-49	⩾ 50 years
Problem Drinking	8*	25	25	25	—	25	—	—	—
Alcoholism (& Gamma-prealcoholism)	87*	17	34	17	18	6	5	1	1

* "Not applicable" responses were omitted (16 problem drinkers, 44 alcoholics, all 28 abstainers and all 41 social drinkers).

It can be seen in *table 3.19.* that *median age at onset of psychological dependence* hardly differs between problem drinkers and (gamma-pre-)alcoholics: this median value is between 20-24 years and 25-29 years.

3.3.4.2. Frequency of drinking because of psychological dependence.

Table 3.20. shows the distribution of *frequency of drinking because of psychological dependence.*

Table 3.20.

Frequency of drinking because of psychological dependence (variable no. 60) in percent.

Diagnostic category	N	Frequency of drinking because of psychological dependence				
		Once yearly or less	1-6 times yearly	Once monthly	Once weekly	Daily
Problem Drinking	9*	11	33	22	33	—
Alcoholism (& Gamma-prealcoholism)	86*	6	20	21	37	16

* "Not applicable" responses were omitted (1 problem drinker, 1 alcoholic, all 28 abstainers and all 41 social drinkers).

* "Never" responses were also omitted (14 problem drinkers, 44 alcoholics).

The results shown in *table 3 20.* indicate, that frequency of escape drinking (drinking because of psychological dependence) increases on the alcoholismic gradient. This finding is not surprising, since psychological dependence (variable no. 58) was used in the construction of the factors "alcoholism" and "problem drinking" (alcoholismic gradient). It does indicate, however, that alcoholics are the "escape drinkers" or "problem drinkers" *par excellence.*

3.3.5. Age at first blackout.

Table 3.21. shows the distribution of age at first blackout.

Table 3 21
Age at first blackout (variable no 68) in percent

Diagnostic category	N	Age at first blackout							
		15-19	20-24	25-29	30-34	35-39	40-44	45-49	⩾ 50
Problem Drinking	12*	17	25	25	25	8	—	—	—
Alcoholism (& Gamma-prealcoholism)	88*	7	24	19	22	11	6	5	7

* "Not applicable" responses were omitted (12 problem drinkers, 43 alcoholics, all 28 abstainers, and all 41 social drinkers)

Table 3.21. shows that *median age at first blackout* hardly differs between problem drinkers and (gamma-pre-)alcoholics: this median value is between 25-29 years and 30-34 years. This finding implies, that *blackout* seems to appear rather *late* in the development of alcoholism and problem drinking, and sharply contrasts Jellinek's statement, that blackout would constitute an *early* sign of impending alcoholism (Jellinek, 1962; Van Epen, 1974). It is not surprising to find blackout among problem drinkers and (gamma-pre-)alcoholics, since variable no. 67 ("frequency of blackout") was used in the construction of the factors "alcoholism" and "problem drinking" (cf. Chapter 2).

3.3.6. Age at inception of loss-of-control.

Table 3.22. shows the distribution of *age at inception of loss-of-control.*

Table 3.22. shows *median age at inception of loss-of-control* differs substantially between gamma-prealcoholics and alcoholics. For gamma-prealcoholics this median value is between 20-24 years and 25-29 years.

Table 3.22
Age at inception of loss-of-control (variable no. 63) in percent.

Diagnostic category	N	Age at inception of loss-of-control							
		15-19	20-24	25-29	30-34	35-39	40-44	45-49	⩾ 50
Gamma-prealcoholism	5	—	80	—	—	20	—	—	—
Alcoholism	97*	5	21	27	18	14	10	3	2

* "Not applicable" responses were omitted (29 alcoholics, all 28 abstainers, all 41 social drinkers, and all 24 problem drinkers).

For alcoholics this median value is between 25-29 years and 30-34 years. Thus, *loss-of-control* seems to appear *later* in alcoholics than in gamma-prealcoholics. It is not surprising to find "loss-of-control" among gamma-prealcoholics and alcoholics, since variable no. 62 ("frequency of loss-of-control") was used in the construction of the factor "alcoholism" (cf. Chapter 2).

Loss-of-control appears to be a rather early symptom among gamma-prealcoholics, which is not surprising since it defines gamma-prealcoholism. It appears for the first time in *47%* of alcoholics *after the age of 30,* and is *absent* in 23% of alcoholics. The differences found, must be viewed against the background of the small number of gamma-prealcoholics.

According to the definitions proposed in this study:

1) The onset of loss-of-control without signs of physical dependence implies the onset of *gamma-prealcoholism, not* of alcoholism as stated by Jellinek (1962).
2) Only the onset of physical dependence (full-blown) indicates the onset of alcoholism i.e. alcohol addiction.
3) The onset of "unconvincing" physical dependence without loss-of-control indicates the onset of prealcoholism.

Nevertheless, the onset of loss-of-control implies the shift from problem drinking to a prealcoholic state (in the sense of gamma-prealcoholism): a step further on the alcoholismic gradient.

3.3.7. Phaseology of some alcoholismic symptoms.

Fig. 3.5. shows phaseology of drinking among social drinkers, problem drinkers and alcoholics and gamma-prealcoholics, i.e. the *median* age at: first drink; first intoxication; onset of psychological

dependence; first blackout; onset of loss-of-control. For purposes of graphic presentation alcoholics and gamma-prealcoholics were grouped together.

Figure 3.5.

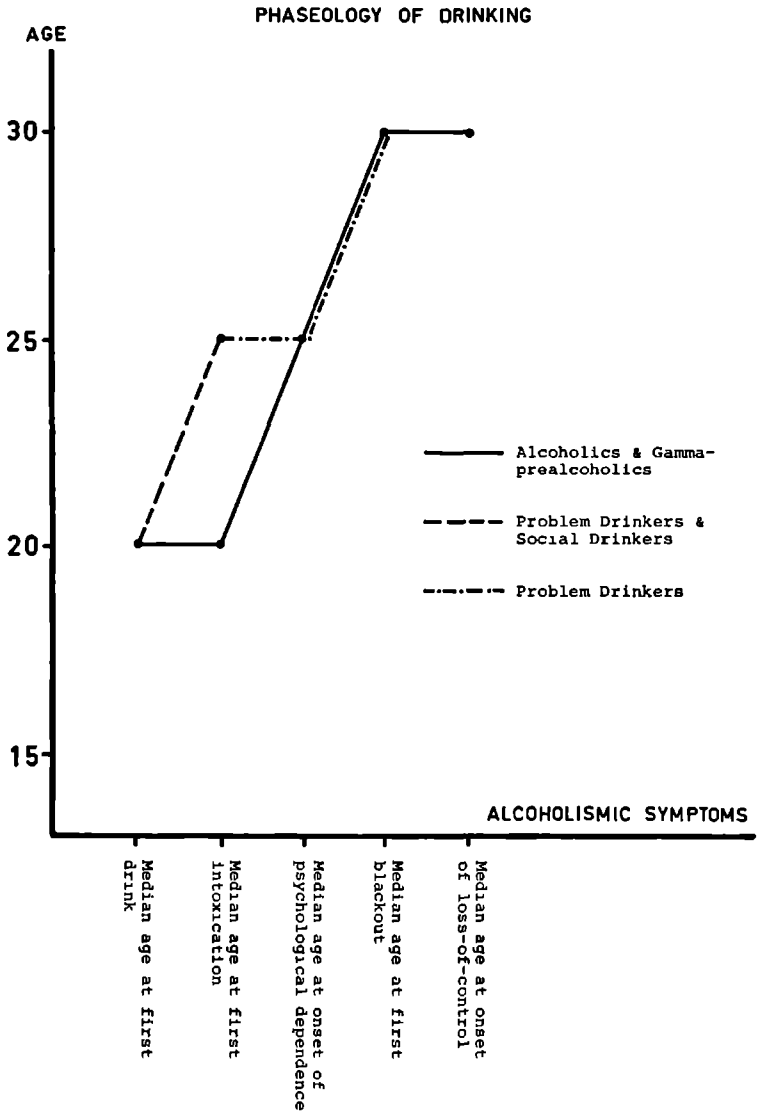


Table 3.23. shows the distribution of decreased, equal and increased tolerance.

Table 3 23.

Distribution of tolerance decreased,equal or increased (variable no 44) in percent, in survey B & C (N = 224)

Diagnostic category	N	Tolerance			
		Decreased (formerly drunk more)	Equal (formerly drunk equally)	Increased (formerly drunk less)	Not applicable
Abstinence	28	0	0	0	100
Social Drinking	41	15	22	12	51
Problem Drinking	24	38	42	17	4
Alcoholism (& Gamma-prealcoholism)	131	55	8	36	2

The results in *table 3.23.* indicate that on the alcoholismic gradient there are *increasing* prevalences of:

a) decreased tolerance;
b) increased tolerance.

Alcoholics are known to have an increased tolerance to ethanol, and increased tolerance is even considered as a major criterion for the diagnosis of alcoholism (N.C.A., 1972; Walsh, 1973).

As shown in *table 3.23.*, however, "increased tolerance" occurs in all drinkers: *36%* of alcoholics and gamma-prealcoholics; *17%* of problem drinkers; *12%* of social drinkers. This seems to be in agreement with Walsh's statement, that tolerance develops quite rapidly, and is at variance with the opinion that tolerance only develops after many years of alcohol ingestion by the alcoholic (Walsh, 1973). Variable no. 44 ("tolerance") was therefore excluded from the set of variables used to construct the factors "alcoholism" and "problem drinking" (= alcoholismic gradient). Other considerations in this decision were:

1) the "tolerance" phenomenon is not adequately covered by variable no. 44; it can be argued that a question like "Can you drink more than half a litre of rhum/whisky/brandy/liqueur/gin *or*

more than 1½ litres of wine *or* 16 bottles/cans of beer without becoming intoxicated?" would better cover this symptom;

2) *decreasing* tolerance is found in *chronic* alcoholism (Jellinek, 1952, 1962).

3.5. AGE

Table 3.24 shows the distribution of age groups in survey B & C.

Table 3.24

Age groups (variable no 2-3) per diagnostic category in survey B & C (N = 224) in percent, mean ages ± S E M

Diagnostic category	N	Age groups						Mean age ± S E M
		15-19	20-29	30-39	40-49	50-59	⩾ 60 years	
Abstinence	28	61	18	4	4	4	10	27 4 ± 3 7
Social Drinking	41	22	27	17	20	7	7	32 9 ± 2 2
Problem Drinking	24	4	38	21	17	13	8	37 5 ± 3 1
Alcoholism (& Gamma-prealcoholism)	131	1	5	35	31	17	12	43 3 ± 0 9

Table 3.25. shows the distribution of age groups in survey A.

Table 3.25

Age groups (variable no 2-3) per diagnostic category in survey A (N = 708) in percent, mean ages ± S E M

Diagnostic category	N	Age groups						Mean age ± S E M
		15-19	20-29	30-39	40-49	50-59	⩾ 60 years	
Abstinence	181	33	21	9	12	9	17	34 9 ± 1 5
Social Drinking	371	19	29	22	14	7	8	33 4 ± 0 8
Problem Drinking	83	13	29	25	16	8	8	35 6 ± 1 7
Prealcoholism	6	0	50	17	17	17	0	35 0 ± 4 3
Gamma-prealcoholism	16	6	38	19	25	13	0	34 7 ± 2 9
Alcoholism	51	10	25	18	20	14	14	39 2 ± 2 1
Total	708	21	27	19	14	8	11	34 5 ± 0 6

The figures in *tables 3.24.* and *3.25* show that:

a) *Abstinence* is concentrated in the *younger* (15-29 years) and *older* age groups (≥ 60 years);
b) *Social drinking* is concentrated between 15-29 years;
c) *Problem drinking* is concentrated above 30 years;
d) *Alcoholism, gamma-prealcoholism* and *prealcoholism* are concentrated in the age groups above 30 years;
e) *Drinking* (non-abstinence) is concentrated between 20-59 years;
f) *Alcoholism* alone is concentrated above 40 years.

These results are in concordance with previously reported age distribution among alcoholics in Aruba (Wever, 1971, 1975), but at variance with findings by others, i.e. correlation of the alcoholismic gradient not with younger but older age groups up to approx. age 50 (Cahalan & Cisin, 1968; Cahalan et al., 1969; Gadourek, 1963; Knupfer & Room, 1964). Excessive drinking was found to correlate with younger age groups in males (Cahalan & Cisin, 1968; Cahalan et al., 1969; Gadourek, 1963; Knupfer & Room, 1964). Gadourek gave as an explanation, that younger males would tend to drink (excessively) to symbolize adult life and that later in life this need would no longer be so pressing (Gadourek, 1963).

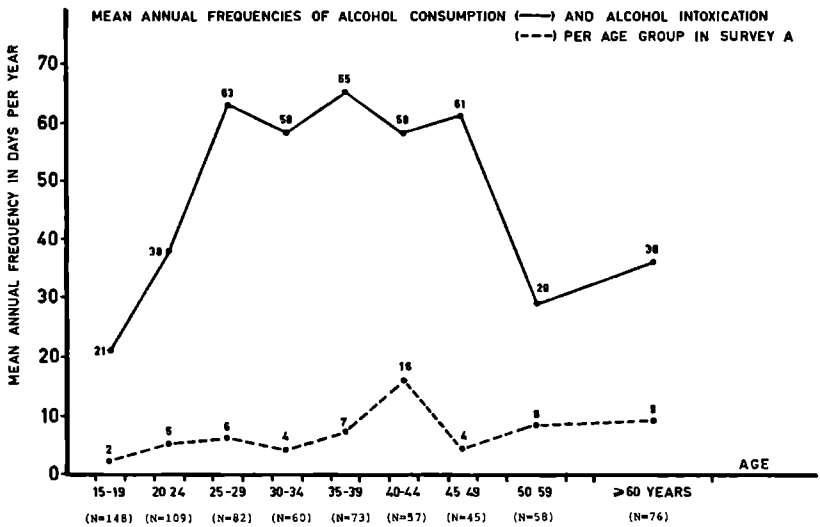
Theoretically, the factor *age* can be interpreted as:
1) *A late sociocultural factor in the microclimate* in the sense of (excessive) drinking to symbolize *adult life* (cf. Chapter 4);
2) A reflection of the *average time* required to develop social drinking, problem drinking and alcoholism;
3) A reflection of increasing life stress up to the age of 50 years and decreasing thereafter, and hence escape drinking around the life stress peak.

The results indicate two trends:
a) An *increase* on the alcoholismic gradient after age 20;
b) A *decrease* on the alcoholismic gradient after age 50.

These two trends agree with the findings of others (Cahalan & Cisin, 1968; Cahalan et al., 1969; Mulford, 1963; Vogel-Sprott, 1974), and are shown in *fig. 3.6.*

As shown in *fig. 3.6.*, there is but a slight correlation between age and mean frequency of alcohol *intoxication*, in contrast to the marked correlation between age and mean frequency of alcohol *consumption.*

Figure 3.6



Though the shape of *fig 3.6.* is not quite curvilinear, it is still suggestive of a possibly curvilinear relationship between age and annual frequency of alcohol consumption (Vogel-Sprott, 1974).

Cahalan et al. (1969) make mention of the following possible explanations for this type of correlation between age and excessive drinking:

a) Older people may have more unpleasant physical reactions to alcohol (metabolic differences) and hence may decrease their drinking frequency;
b) Older people may feel less need of alcohol to relieve tension, either because they are under less strain or have turned to tranquillizers or other coping mechanisms (Knupfer & Room, 1964);
c) Middle-aged couples may have more money and time to enjoy drinking at leisure, and hence increase their drinking frequency;
d) Generational differences in drinking frequency i.e. older people may drink less, because they grew up at a time when drinking was less widespread and hence retain their old habits;
e) Higher mortality among alcoholics (Schmidt & De Lint, 1972) and heavy drinkers would reduce the prevalences of these pathological drinkers among the older age groups;

f) Older people may be more concerned about their health and hence may reduce their drinking for this reason (Knupfer & Room, 1964).

Knupfer et al. (1964) stated that older people possibly are less active, less impulsive, more dignified, and more stable than younger people, all of which characteristics would lead to greater restraint in drinking.

3.6. PUBLIC OPINIONS

3.6.1. Opinions concerning the alcoholic.

Table 3.26. shows the distribution of *opinions concerning the alcoholic.*

Table 3.26.

Opinions concerning the alcoholic (variable no. 155) in percent, in survey B & C (N = 224).

Diagnostic category	N	Opinions concerning the alcoholic The alcoholic considered as:					
		An emotionally disturbed person	A socially degraded person	A morally degenerated person	A person lacking willpower	A sick patient	Not appl.
Abstinence	28	4	0	11	57	29	0
Social Drinking	41	5	7	2	61	24	0
Problem Drinking	24	13	4	4	46	29	4
Alcoholism (& Gamma-prealcoholism)	131	4	12	7	39	35	4

Though the differences in *table 3.26.* are not statistically significant, there is a slight trend indicating that with increasing alcoholismic gradient also the prevalence of "the alcoholic is a sick patient" responses increases, probably reflecting indoctrination by AA.

In *survey A* (N = 708) this same trend is discernible, but again, the differences were not statistically significant. 32% of all respondents in survey B & C, and 42% in survey A responded with "the alcoholic is a sick patient" responses or "the alcoholic is an emotio-

nally disturbed person" responses. This implies that there is but poor acceptance of the *disease concept of alcoholism* (Jellinek, 1960) in Aruba.

3.6.2. Opinions concerning AA work.

Table 3.27. shows the distribution of *opinions concerning AA work.*

The results as shown in *table 3.27.* show two trends:

a) With increasing alcoholismic gradient the "AA has reasonably much success" responses also increase;
b) With increasing alcoholismic gradient the "AA is unknown to me" responses decrease.

Table 3.27.

Opinions concerning AA work (variable no. 115) in percent, in survey B & C (N = 224).

Diagnostic category	N	Opinions concerning AA work						
		Unknown to me	Does not interest me	AA makes men unmanly	AA has no succes at all	AA has only moderate succes	AA has reasonably much success	Not. appl.
Abstinence	28	57	4	0	0	25	14	0
Social Drinking	41	34	0	0	5	17	39	5
Problem Drinking	24	42	4	0	4	29	21	0
Alcoholism (& Gamma-prealcoholism)	131	5	4	0	0	12	79	0

Both trends can be considered as a result of indoctrination by AA.

In *survey A* (N = 708) these both trends are discernible, but the differences were *not* statistically significant.

Moreover, the results indicate, that overall 73% of respondents in survey B & C and 55% in survey A responded favorably about AA work success, i.e. with "AA has moderate success" or "AA has much success" responses. It may be concluded, that in general, public opinion is positive toward AA.

3.7. CONCLUSIONS

1) Frequencies of alcohol consumption and alcohol intoxication clearly increase in the categories abstinence, social drinking, problem drinking, prealcoholism, gamma-prealcoholism and alcoholism (alcoholismic gradient). This was shown both in survey A and in survey B & C.
2) Volumes of alcohol consumption also clearly increase on the alcoholismic gradient.
3) The lower limit value of 150 ml. as lowest mean daily alcohol intake by alcoholics is questionable. Except this study, there are more reports which fail to confirm this lower limit value (Lereboullet, 1964; Lundquist, 1972).
4) Familial monthly expenses on beverage alcohol clearly increase on the alcoholismic gradient.
5) Alcoholics, gamma-prealcoholics and prealcoholics in Aruba tend to prefer *hard liquor*, while problem drinkers and social drinkers prefer *beer*. This divergence in beverage preference is at variance with other studies (Cahalan et al., 1969; De Lint & Schmidt, 1971, 1971a, 1971b).
6) Alcoholics and gamma-prealcoholics tend to prefer the cheaper types of hard liquor.
7) Drinking bouts tend to grow longer with increasing alcoholismic gradient.
8) Excessive drinking and excessive smoking during weekends clearly increase on the alcoholismic gradient.
9) Excessive drinking on cultural, national and religious occasions (cultural crisis of alcohol consumption) clearly increases on the alcoholismic gradient.
10) Age at first drink differs little if nothing among the diagnostic categories.
11) Age at first intoxication is significantly *lower* for alcoholics and gamma-prealcoholics as compared to the other categories.
12) Age at onset of psychological dependence hardly differs among problem drinkers and alcoholics & gamma-prealcoholics.
13) Frequency of drinking because of psychological dependence increases on the alcoholismic gradient: alcoholics are the "problem drinkers" *par excellence*.

14) Since both frequencies and volumes of alcohol consumption increase on the alcoholismic gradient, alcoholics can be considered as the excessive drinkers *par excellence.*
15) Age at first blackout differs little or nothing among problem drinkers and alcoholics & gamma-prealcoholics.
16) Age at inception of loss-of-control is significantly *lower* for gamma-prealcoholics as compared to alcoholics.
17) Tolerance as evaluated in this study, does not significantly differentiate the diagnostic categories. This may possibly be due to the fact that this dimension was inadequately operationalized in this study.
18) Especially in survey B & C there are significant age differences between the diagnostic categories.
19) Frequencies of alcohol consumption increase with age up to 45-49 years, but decrease after age 50.
20) Frequencies of alcohol intoxication show less impressive changes with age; there is a peak at 40-44 years, while at the remaining age groups the values vary very little.
21) There is but poor acceptance of the "disease concept of alcoholism" in Aruba.
22) General public opinion in Aruba is positive to Alcoholics Anonymous.

ANALYTICAL EPIDEMIOLOGY: PREDISPOSING AND PERPETUATING FACTORS IN ALCOHOLISM AND ITS PRECURSOR STAGES. THEORETICAL ORIENTATION

4.1. ETIOLOGY OF ALCOHOLISM

Among the many theories concerning the etiology of alcoholism, there is not a single one capable of solely explaining it (Albrecht, 1973; Beaubrun, 1969, 1971; Canavan, 1971; Cork, 1969; Fox, 1971).

Therefore the etiology of alcoholism can best be viewed from a multifactorial standpoint (Albrecht, 1973; Cork, 1969; Edwards, 1970; Wallace, 1972). Basically, this study's view of the etiology of alcoholism is in agreement with Albrecht's assumption: "Biological potentialities consist of physiological states which are genetically determined, but the transformation of these physiological states into behavioral characteristics is a function of social and cultural conditioning" (Albrecht, 1973).

4.1.1. Hypothetical model of the etiology of alcoholism.

Before dealing in detail with the various etiological factors - as shown in table 4.1. - *a hypothetical model* of the etiology of alcoholism will be proposed, to serve as a framework against which the evidence to be presented may be evaluated. The hypothetical model, based on data from the literature, is the following: While the majority of people older than 15 years do drink alcoholic beverages, only a relatively small percentage of them drink excessively. Those who do drink excessively, do this:

a) Because of genetic predispositions leading to excessive alcohol consumption and/or to the development of physical dependence to alcohol (Albrecht, 1973; Jellinek, 1960, 1962).
b) Because they need something which *quickly* anesthetizes mental distress, such as for instance anxiety, depression, frustration, loneliness, feelings of insecurity (Fox, 1967, 1968; Milt, 1969; Wallerstein, 1968); this mental distress may arise from conscious (e.g. marital conflicts) or from unconscious problems in daily

life (e.g. latent homosexuality, dependency needs), from conscious or unconscious conflicts stemming from early youth experiences (e.g. interparental disharmony, paternal punitiveness, broken homes) (Edwards, 1970; Esser, 1965; Gadourek, 1963; Jones, 1966; Milt, 1969; Wahl, 1956); this need of quick relief from mental distress develops into *habituation* through *conditioned reflex learning* (Conger, 1956; Jellinek, 1960; Kalant & Kalant, 1971; Keller, 1972; Milt, 1969; Smart, 1965; Wexberg, 1951).

c) Because of socially, culturally or economically determined factors, facilitating alcohol consumption, such as low price of beverage alcohol, a country's tolerant attitude towards heavy drinking and intoxication, especially for the purpose of obtaining quick relief from mental distress or for the mere purpose of obtaining pleasure (Albrecht, 1973; Milt, 1969).

With regard to *mental distress* it can be stated:

1) that in many instances the original noxious factor derives from outside the affected individual (e.g. interparental conflicts in youth);
2) that these external factors are *internalized* by the affected individual;
3) that the burden of the original noxious factor will only lead to decompensation either when this burden is too heavy as compared to the individual's bearing capacity, or when the individual's bearing capacity is insufficient as compared to the "weight" of the burden; this correlates with the postulated lowered frustration threshold (Jellinek, 1960).

Concerning the *conditioned reflex learning*, it can be stated, that the pattern: distress → alcohol → relief from distress, is frequently repeated and becomes overlearned; the reinforcement (relief from mental distress) is immediate in contrast with the delayed reinforcement of other actions for handling distress; the conditioned reflex progressively preempts the field of the response choices, which field narrows rapidly down to virtually this one response to the widest range of stimuli. The formation of this conditioned reflex or habituation has been called "psychological addiction" or a "mechanism with functional autonomy" (Wexberg, 1951); Jellinek has proposed for this mechanism the term *psychological dependence* (Jellinek,

1960) to be distinguished from *physical dependence* or *addiction* (cf. Chapter 1).

This hypothetical model is congruent with the genetic, psychological and sociocultural & economic factors as shown in *table 4.1.*

Table 4.1. gives a review of commonly mentioned etiological factors.

Table 4.1.
Etiological factors in alcoholism.

1) Genetic factors;	
2) Somatic factors:	a) Nutritional deficiencies; b) Endocrinopathies;
3) Psychological factors:	a) Genuine psychiatric diseases; b) Psychological factors other than psychiatric diseases;
4) Social, cultural & economic factors:	a) In microclimate; b) In macroclimate.

The "*masculine façade*" hypothesis (Mc Cord & Mc Cord, 1962) could not be tested in this study, since it is a retrospective study, not a longitudinal one, like Mc Cord's. Mc Cord's hypothesis is based on the observation, that there are marked differences in behavior between alcoholic males in adult life and "prealcoholic" boys in childhood: "prealcoholic" boys manifest a façade of intense masculinity (aggression, hyperactivity, independence, self-confidence), as contrasted by the alcoholic's behavior in adulthood (passivity, open expression of dependency needs, lack of self-confidence). Mc Cord assumes, that the truly "alcoholic" behavior is *suppressed* in childhood, and that later in adult life, once the disease of alcoholism has set in, the person's defenses collapse and subsequently this latent "alcoholic" behavior emerges. Alcoholics in childhood are often subjected to experiences which one would expect to lead to intensified dependency needs and to confusion in self-images: e.g. frequent interparental conflicts or the absence of a responsible identification model in early childhood. One way of handling these heightened dependency needs would be to *repress* them. One way of dealing with the absence of a responsible male identification model would be to accept the culturally-sanctioned image of masculine behavior. By asserting his "masculinity" this type of person could temporarily

overcome his basic confusion. However, his dependency needs continue to plague him, yet his "masculine façade" leaves little room for their satisfaction. At some point in adulthood, this type of person learns that alcohol may provide a compromise solution to his dilemma. Through heavy drinking, widely regarded as virile behavior, he may succeed in simultaneously satisfying his dependency needs and maintaining his precarious grip on a masculine self-image (Mc Cord & Mc Cord, 1962). Of course, this hypothesis is only applicable to the development of *male* alcoholism.

Table 4.1. is merely intended to give a general outline of possible etiological factors. The present study deals mainly with the sociocultural, economic and psychological factors as mentioned above, and to a lesser extent with presumably genetic factors.

4.1.2. Genetic factors.

Genetic factors can be considered as *predisposing*, even as causative factors, whereas sociocultural and psychological factors include both *predisposing* and *perpetuating* factors. Predisposing and perpetuating factors will be defined below.

Human traits are not exclusively determined by genetics *or* environmental factors, but by both in concerted action. This is especially true for *continuously* distributed traits (body weight and height, various psychic attributes of man) as opposed to *dichotomous* traits (e.g. phenylketonuria). For continuously distributed traits it has been shown that statistical results of correlations among relatives were exactly as expected if a hereditary basis was assumed consisting of many genes, each exerting a small effect on the trait in question; this is the so-called *polygenic model* (Mc Clearn, 1973). Such a model *may* be applicable for alcoholism. A key concept of this model is *heritability*, which expresses the proportion of the total variability in a population ascribable to genetic variation among the individuals of that population. In formula: $H = V_G / (V_G + V_E)$.

In this formula H is heritability; V_G is the genetic variance component; V_E is the environmental variance component. The formula implies that *heritability* decreases with increasing environmental variance, and increases with decreasing environmental variance or increasing genetic variance. This model is also appropriate as an antidote to the misconception, that to admit a genetic involvement in a

disease would be a counsel of despair, since it demonstrates that the *phenotype* may be altered by modifying environmental factors (Mc Clearn, 1973).

4.1.2.1. Genetic factors in autochthonous Arubans.

It has been stated earlier, that alcoholism prevalence rates among Arubans are higher than among the inhabitants of the five other islands of the Netherlands Antilles, i.e. Bonaire, Curaçao, St. Martin, St. Eustatius and Saba (Berkley, 1956, 1957; Bosch, 1836; Steenmeyer, 1957; Wever, 1971, 1975).

4.1.2.1.1. Amerindian genetic factors.

The higher prevalence rates of alcoholism among Arubans for many decades have been ascribed to "the Indian blood" of Arubans, a view which has been repeated recently (Bijl, 1971). It is a frequent observation, that alcoholism prevalence rates are high among Amerindians (Caravedo, 1957; Dozier, 1966).

Still, it has not been proven that this is due to genetic factors.

Against this hypothesis can be argued that among Chinese in the U.S.A. - and both Amerindians and Chinese belong to the Mongolian race - prevalence of alcoholism is *low* (Chu, 1972). Moreover, this rather simplistic (pseudo?)genetic theory can be considered to reflect sociocultural factors as well, since the higher prevalence of alcoholism in the original Aruban population, with its high prevalences of both Amerindian genes and Amerindian cultural heritage, can be caused by the transfer of sociocultural factors existing in the original Amerindian population or in the trihybrid Amerindian-Caucasoid-Negroid population of the 16th through 19th century.

4.1.2.1.2. Genetic drift and inbreeding.

It can also be argued that the high(er) prevalences of alcoholism among the Aruban population may not be due to specifically Amerindian genetic factors, but to other genetic phenomena such as *genetic drift* and *inbreeding*, both frequently occurring in closed communities like Aruba was for centuries until the nineteen-twenties. Both these factors promote the appearance of (particularly recessive) genetic properties out of *any* of the racial origins of a polyhybrid population.

4.1.2.2. The X-linkage hypothesis; association of alcoholism with the deuteranopsia type of color-blindness.

This hypothesis was proposed by Cruz-Coke & Varela (Cruz-Coke & Varela, 1966). It implies, that alcoholism would be "a component of a genetic polymorphism of the color-vision supergenes located on the X-chromosome". In other words: a genetically determined deficiency of the enzyme alcohol dehydrogenase would provoke both alcoholism and deuteranopsia since this enzyme is involved in the rhodopsin/vitamin A cycle. The relative frequency of male and female alcoholism prevalence rates was seen as consistent with the hypothesis that predisposition to alcoholism would be an X-linked recessive trait. It was furthermore postulated that if sensitive tests were used, deuteranopsia should be demonstrable in the heterozygotes, although in classical genetic theory female carriers of X-linked recessive traits do not reveal abnormalities. This hypothesis received some confirmation (Sassoon & White, 1970) and much criticism:

1) a disturbance in the rhodopsin/vitamin A cycle would lead to a general impairment of vision rather than to a specific color-vision defect (Gorrel, 1967);
2) in color-blindness no dehydrogenase enzymes are implicated (Gorrel, 1967);
3) failure to confirm the Chilean findings (Thuline, 1967);
4) defective color-vision tests originally found in acutely ill patients, returned to normal as the sensorium cleared (Thuline, 1967);
5) in alcoholism there is striking resemblance in drinking practices between fathers and sons, which *cannot* be adequately explained on the basis of X-linkage (Mc Clearn, 1973).

4.1.2.3. Blood groups; association of alcoholism with blood group A.

This hypothesis was proposed by Nordmo (Nordmo, 1959), but was not confirmed in other studies (Camps & Dodd, 1967).

4.1.2.4. Family studies; association of alcoholism with parental alcoholism, alcoholism in siblings and in extended family, and non-specific hereditary predispositions.

4.1.2.4.1. Non-specific hereditary predispositions.

Jellinek carried out an extensive review of the heredity of alco-

holics, and concluded, that about 35% showed "hereditary taint": a family history of any kind of psychosis or psychopathy *excluding* inebriety of parents (Jellinek, 1945).

Ämark found high prevalences of psychogenic psychosis and psychopathy among parents and siblings of *alcoholics* (Ämark, 1951).

4.1.2.4.2. Parental alcoholism, alcoholism in siblings and extended family.

High prevalences of alcoholism in parents, siblings and other relatives of alcoholics have been found (Ämark, 1951; Fox, 1968; Van der Does de Willebois, 1965). However, these family studies may merely reflect sociocultural factors predisposing to alcoholism instead of genetic factors, in the sense of early environmental moments conducive to the "alcoholismic learning process" (Jellinek, 1960; Kalant & Kalant, 1971; Keller, 1972), such as the parental example of inebriety.

4.1.2.4.3. Twin studies.

Twin studies have been comprehensively reviewed by Edwards and Mc Clearn (Edwards, 1970; Mc Clearn, 1973). Twin studies rather than family studies in assessing the problem of alcoholism inheritance were conducted extensively by Partanen et al. (Partanen et al., 1966). It was found, that higher percentages of *monozygotic* twins than dizygotic twins had a drinking problem, and that hereditary factors affected the (excessive) use of alcohol but *not* its social consequences. Through multivariate analysis three dimensions were identified: *density* (frequency of consumption); *amount* (the quantity typically consumed on single occasions); *loss-of-control* (Partanen et al., 1966). For *density* and *amount* genetic influence was found to be quite large; for *loss-of-control* there was insufficient evidence to establish a hereditary component (Mc Clearn, 1973; Partanen et al., 1966).

4.1.2.4.4. Studies with adopted children.

An adopted child shares environmental factors given by a family but *not* its genetic lineage. No support for a genetic view has been reported by Roe's study (Roe, 1944).

4.1.2.4.5. Studies with half-sibs.

The study by Schuckitt et al. (1972) as reviewed by Mc Clearn (Mc Clearn, 1973) gave good support for the genetic view, since it was found that biological parentage was more related to subsequent alcoholism than the rearing parentage.

4.1.2.5. Sex limitation.

It can be postulated, that alcoholism would be located on autosomes in which the phenotypic expression is preponderantly limited to male sex. The possible role of such a factor in alcoholism cannot be excluded on the basis of present knowledge (Mc Clearn, 1973). In this respect, a genetic model can provide a more precise and systematic explanation than can a sociocultural model since only a minority of siblings (and especially brothers) of alcoholics also become alcoholics *in spite of sharing* with their alcoholic sib a great deal of environmental factors. And if affected, there is great preponderance of *brothers* affected (Mc Clearn, 1973).

4.1.2.6. Animal studies.

Research on animals has been comprehensively reviewed by Edwards and Mc Clearn (Edwards, 1970; Mc Clearn, 1973). The main fact that emerges from these investigations is that, with a number of species, strains can be bred, which exhibit a heightened appetite for alcohol as judged by behavior in a free-choice experimental situation. Nevertheless, it is important to realize that experimental animal alcoholism is not identical with human alcoholism, although results in animal studies may have some (remote) bearing on humans.

4.1.3. Somatic factors.

The principal theories concerning these factors deal with:

1) Nutritional deficiencies.
2) Endocrinopathies.

4.1.3.1. Nutritional deficiencies.

— *Mardones* stated, that alcoholism may be rooted in nutritional deficiencies such as *thiamine (vitamin B_1) deficiency* and/or *deficiency of a (hypothetical) specific factor N_1* in the diet; these

deficiencies in animal experimental work led to increased alcohol consumption, and were considered as *acquired* deficiencies (Mardones, 1951).

— *Williams* stated, that alcoholism could be considered as a "*genetotrophic disease*" i.e. an *inherited pattern of individual metabolic peculiarities which cause an increased requirement of vitamins and other substances which are operative in intermediary metabolism.* These increased requirements were considered to reflect both congenital and acquired deficiencies (Williams, 1947).

These nutritional theories might have some bearing on the development of alcoholism in Aruba, since deficiencies of vitamins B and C have been found in the nutrition of Arubans (Steenmeyer, 1957). It should be remembered however, that nutritional deficiencies especially those mentioned above, frequently occur as *consequences* of the well-known deficient dietary customs of alcoholics (Mardones, 1951; Williams, 1947).

4.1.3.2. Endocrinopathies.

— *Smith* stated, that alcoholism may imply a constellation of *pituitary-adrenal* and *gonadal dysfunctions* pre-existent to alcoholism, which would set up a demand for alcohol (Smith, 1949).

— *Tintera & Lovell* stated, that *hypoadrenocorticism* pre-existent to alcoholism in some alcoholics and acquired by others, would set up a demand for alcohol (Tintera & Lovell, 1949).

These endocrinological theories seem to fit well with *Selye*'s findings concerning decreased stress tolerance in psychosomatic disease (Selye, 1946). *Merry* has shown, however, that hypoadrenocorticism and hypofunction of the hypothalamic-pituitary-adrenocortical axis rather are *consequences* than causes of alcoholism (Merry, 1971a; Merry & Marks, 1969).

4.1.4. Psychological factors.

These psychological factors can be divided into:

1) Genuine psychiatric diseases;
2) Psychological factors other than genuine psychiatric diseases.

4.1.4.1. Psychiatric diseases.

Patients with "premorbid" psychological properties would have a

higher risk of developing alcoholism. In this category the following congenital psychiatric diseases can be mentioned: oligophrenia, endogenous manic depressive psychosis, endogenous vital depression, degenerative hysteria, schizophrenia, degenerative psychopathy, and epilepsy (Esser, 1967; Jellinek, 1960). In this category also the following asquired psychiatric diseases can be mentioned: reactive depression, psychogenic psychosis and neurotic diseases, especially anxiety neurosis, obsessive-compulsory neurosis and hysteric neurosis (Esser, 1967; Jellinek, 1960; Van der Does de Willebois, 1965).

4.1.4.2. Psychological factors other than genuine psychiatric diseases.
These factors are shown in *table 4.2.*

Table 4.2
Psychological factors other than psychiatric diseases

1) *Fixation at or regression to various levels of psychosexual development* (oral, anal, or phallic-oedipal fixation or regression) (Gadourek, 1963, Milt, 1969).
2) *Anxiety* (Jellinek, 1960, Horton, 1943, Sayres, 1956, Wanberg & Knapp, 1970)
3) *Boredom* (Van der Does de Willebois, 1965)
4) *Solitariness and low social participation* (Cork, 1969, Gadourek, 1963)
5) *Escapism* (Cahalan et al., 1969; Edwards, 1970, Gadourek, 1963, Jellinek, 1960, Kalant & Kalant, 1971)
6) *Suicidality* (Gadourek, 1963, Milt, 1969, Palola et al., 1962)
7) *Hedonism* (Gadourek, 1963, Jellinek, 1960, Milt, 1969)
8) *Introversion and schizoid traits* (Esser, 1967, Jellinek, 1960, Wanberg & Knapp, 1970, Wexberg, 1951).
9) *Psycholability* (Esser, 1967, Jellinek, 1960).
10) *Dissatisfaction* (Gadourek, 1963)
11) *Low frustration threshold* (Jellinek, 1960).
12) *Reinforcement theory of learning* (Jellinek, 1960, Kalant & Kalant, 1971; Keller, 1972)

4.1.5. Social, cultural and economic factors.
These can be divided into:
1) Factors in the *micro*climate;
2) Factors in the *macro*climate.

4.1.5.1. Factors in the microclimate.
These can be divided into:
1) *Early* factors in the microclimate (before adult life);
2) *Late* factors in the microclimate (during adult life).

The childhood home may predispose to later alcoholism (Åmark,

1951, Cahalan & Cisin, 1968; Cahalan et al., 1969; Cork, 1969, Criteria Committee, 1972; Edwards, 1970; Esser, 1965; Fox, 1968; Jackson, 1962; Jellinek, 1945; Jones, 1966; Krimmel, 1973, Mc Cord & Mc Cord, 1962; Milt, 1969, Robins et al., 1962; Van der Does de Willebois, 1965, Wahl, 1956; Wanberg & Knapp, 1970). Deficient education by parents, deficient family integration, boredom as a passive form of self-alienation, inadequate motherhood, and especially inadequate fatherhood in the sense of an "unreachable father's image" or an "empty, rejectable father's image", have been found as factors conducive to alcoholism (Van der Does de Willebois, 1965)

Table 4 3 shows a number of *early* factors in the microclimate (i.e the primary environment of family and extended family).

Table 4 3

Early factors in the microclimate

1) Non specific psychiatric predispositions (Jellinek, 1945) family history of any psychosis or psychopathy except parental inebriety,
2) Parental & familial alcoholism (Åmark, 1951, Edwards, 1970, Fox, 1968, Van der Does de Willebois, 1965, Wanberg & Knapp, 1970)
   — reinforcement theory of learning (Albrecht, 1973, Bacon, 1973, Jellinek, 1960, Kalant & Kalant, 1971)
   — alcoholismic learning process (Albrecht, 1973, Keller, 1972)
   — early childhood emotional deprivation (Cork, 1969, Edwards, 1970),
3) Inadequate motherhood (Jones, 1966, Mc Cord & Mc Cord, 1962, Milt, 1969, Robins et al, 1962, Van der Does de Willebois, 1965),
4) Inadequate fatherhood in the sense of an 'unreachable father s image" or empty father s image ' (Van der Does de Willebois, 1965),
5) Paternal rejection, paternal punitiveness, paternal escapist reaction to crisis (Cork, 1969, Criteria Committee, 1972, Edwards, 1970, Robins et al, 1962, Wahl, 1956),
6) Deficient family integration (Jones, 1966, Milt, 1969, Robins et al, 1962, Van der Does de Willebois, 1965),
7) Interparental conflicts (Jones, 1966, Mc Cord & Mc Cord, 1962, Milt, 1969, Robins et al, 1962),
8) Deficient education by parents (Jellinek, 1960, Van der Does de Willebois, 1965),
9) Illegitimacy (Edwards, 1970),
10) Incest (Edwards, 1970),
11) Virility complex (Gadourek, 1963, Mc Cord & Mc Cord, 1962) the (excessive) use of alcohol to symbolize manliness and adult life in males,
12) Low social status of parents (Robins et al, 1962),
13) Poverty and poor housing conditions in parental home,
14) Urban domicile (Gadourek, 1963, Wallace, 1972),
15) Deficient school education (Cahalan & Cisin, 1968, Cahalan et al, 1969, Pittman & Gordon, 1962),
16) (Catholic) religion (Gadourek, 1963, Wallace, 1972),
17) Parental absence or loss, broken home in early youth (Cahalan & Cisin, 1968, Cahalan et al, 1969, Wahl, 1956, Wanberg & Knapp, 1970)

Table 4.4. shows a number of *late* factors in the microclimate.

Table 4 4
Late factors in the microclimate

1) Age (Gadourek, 1963) young adults (20-30 years) especially males, would tend to excessive drinking to symbolize adult life,
2) Low social status of drinker,
3) Poverty and poor housing conditions in drinker s home,
4) Marital conflicts (Jones, 1966, Edwards, 1970, Gadourek, 1963, Wanberg & Knapp, 1970),
5) Conflicts with children (Edwards, 1970),
6) Conflicts in extended family (Gadourek, 1963),
7) Anomy (Gadourek, 1963),
8) Authority conflicts,
9) Divorced, widowed or single civil state,
10) Availability of alcohol (De Lint & Schmidt, 1970, 1971, 1971a and 1975, Gadourek, 1963, Wallace, 1972) the relatively low cost of beverage alcohol as compared to individual per capita income (economic availability),
11) Dissatisfaction with life, health, housing conditions, economic situation (Edwards, 1970, Gadourek, 1963)

4.1.5.2. Factors in the macroclimate.

Circumstances in the community at large may predispose to and/or perpetuate alcoholism.

Table 4 5. shows a number of factors in the macroclimate.

Table 4 5
Factors in the macroclimate

1) Social normativity (Albrecht, 1973, Beaubrun, 1971, Jansen, 1969, Gadourek, 1963, Plaut, 1967) predominantly tolerant or ambivalent attitudes towards (excessive) use of alcohol in a community,
2) Availability of alcohol (De Lint & Schmidt, 1970, 1971, 1971a and 1975, Gadourek, 1963, Wallace, 1972)
   — the relatively low cost of beverage alcohol as compared to modal per capita income (economic availability),
   — absence of legal restrictions regarding sales of beverage alcohol (legal availability),
   — presence of numerous geographically well spread bars, restaurants, etc (bar & restaurant availability),
3) High mean annual alcohol consumption (De Lint & Schmidt, 1970, 1971, 1971a and 1975, Ledermann, 1956 and 1964),
4) Social habit forming
   — the learning of alcohol consumption in certain social settings, e g frequency of offering drinks to other people, drinking with working companions, hospitality drinking (Bacon, 1973, Heath, 1962),
5) Cultural crisis
   — the learning of alcohol consumption in certain social settings, e g (excessive) drinking on religious and national festive days, e g drinking to show hospitality (Heath, 1962, Horwitz et al , 1967),
6) Stress in the working situation (Gadourek, 1963, Wanberg & Knapp, 1970),
7) Proneness to social pressure to drink/social dependence
   — drinking under social pressure of friends,
8) Anomy (Gadourek, 1963)

4.2. PREDISPOSING AND PERPETUATING FACTORS

Predisposing and *perpetuating* factors are those factors capable of explaining the shift in the *socio-psycho-biologic gradient:* abstinence → social drinking → problem drinking → prealcoholism or gamma-prealcoholism → alcoholism.

Gordon (1956; cited in Jellinek, 1960) proposed for alcoholism a "biologic gradient", a concept useful in understanding mass disease.

In this study this gradient is denominated a "socio-psycho-biologic gradient" or "*alcoholismic gradient*" since it implies not only biologic factors (withdrawal syndrome, somatic complications of alcoholism) but also social and psychological factors (social and psychological correlates of alcoholism). In this gradient *abstinence* represents - in analogy with infective processes - the "non-infected" yet possibly susceptible part of the population. *Social drinking* represents the part of the population without clinically apparent "infection", but nevertheless "infected". *Problem drinking* represents the part of the population with clinically apparent relatively minor effects of "infection". *Prealcoholism and gamma-prealcoholism* represent the part of the population with clinically apparent yet not classical effects of "infection". *Alcoholism* represent the part of the population with clinically apparent classical effects of "infection".

Of course this comparison with infective diseases does not intend to define alcoholism as an infective process, but merely to point to the analogies between the alcoholismic gradient and the pathways of pathogenesis of infective diseases.

Predisposing factors are defined as those factors which:

a) chronologically precede the shift from abstinence to alcoholism as described above;
b) unequivocally can be considered as *causes,* not as consequences of alcoholism.

The relationship between alcoholism and its predisposing factors is as follows:

Predisposing factors → Alcoholism

Nothwithstanding the fact, that there is widespread agreement among alcoholism researchers, that significant *early* relationships or the lack of these, play an important role in the development of the

personality, there is a surprising paucity of such studies in the literature on alcoholism (Bell et al., 1976; Milt, 1969; Wahl, 1956). The problem of defining really predisposing factors and perpetuating factors, has also been indicated by Mc Cord & Mc Cord (Mc Cord & Mc Cord, 1962).

Perpetuating factors are defined as those factors which:

a) chronologically coincide with the development of the alcoholismic gradient, and hence
b) may be considered both as *causes* and as *consequences* of alcoholism, and
c) when present, may perpetuate the shift on the alcoholismic gradient.

The relationship between alcoholism and its perpetuating factors can be considered to imply *circular causality:*

Perpetuating factors $\rightleftarrows$ Alcoholism

Krimmel has characterized this relationship as a *pathological complementary* relationship (Krimmel, 1973). This implies, that for instance in the marital interaction, the behavior of one partner provokes and presupposes the other partner's reactive behavior, and vice versa (Krimmel, 1973). Generally, it implies, that the action of one part of the pathological complementary relationship presupposes and perpetuates the reaction of the other part(s).

Some characteristics of pathological complementary relationships are the following:

a) There is a *circular quality.* Thus, attempts to break the chain at some arbitrary point, in order to determine the cause or to achieve improvement, are irrelevant if not impossible.
b) There is *increasing rigidity* in the relationships, with fewer alternative patterns available to the partners or constituent parts of the relationship (Krimmel, 1973).

4.2.1. The relationship between fathers and sons as measured with the "Parent Behavior Inventory" (P.B.I.).

Parental attitudes are correlated with the *education* of their children. If the educational situation is defined as parental influence

upon the children during a certain period of their lives, it is possible to characterize parental behavior by a great number of descriptions, e.g.: overprotective parents; demanding parents; indulgent parents; democratic parents; dominant parents; rejecting parents. Many researchworkers have tried to explain deviant behavior in children and adults (e.g. alcoholism, delinquency, neurosis, schizophrenia) through various types of parental behavior in early youth (Heydendael et al., 1972). Symonds (1939; cited by Heydendael et al., 1972) constructed a model consisting of two dimensions: a) *acceptance* versus *rejection;* b) *dominance* versus *submission.*

In a later study more or less identical dimensions were found (Heydendael et al., 1972).

With respect to models of parental behavior, Schaefer's model, as applied in the study of Heydendael et al. (1972) is one of the best elaborated and one of the most frequently applied. Therefore in this study the P.B.I.-questionnaire has been added, in the abbreviated form as proposed by Heydendael et al.

Schaefer's hypothetical model of parental behavior is shown in *fig. 4.1.*

The model in *fig. 4.1.* shows, that parental behavior is based on *two bipolar main dimensions,* namely *acceptance vs. rejection* and *control vs. promotion of autonomy.*

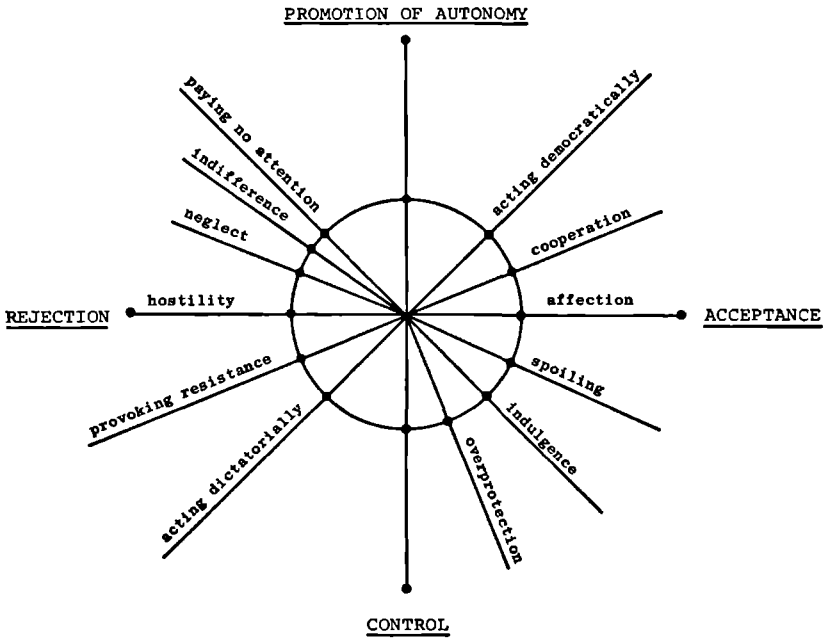
Combination of these two main dimensions leads to *four principal factors:*

a) *acceptance-promotion of autonomy (overindulgence);*
b) *acceptance-control (overprotection);*
c) *rejection-promotion of autonomy (rejection);*
d) *rejection-control (punitiveness).*

Schaefer's model, is only a surveyable simplified expression of the complexity of parental behavior. Schaefer's original P.B.I.-questionnaire consisted of 192 variables with three possible answers per variable; this was reduced to 72 items with four possible answers per item (Heydendael et al., 1972). This condensed P.B.I. was employed in this study, and presented only to a number of *male* respondents to evaluate their early youth experiences with their fathers, since the influence of fathers was considered significant in the development of male alcoholism (Criteria Committee, 1972; Edwards, 1970; Van der

fig. 4.1.

SCHAEFER'S HYPOTHETICAL MODEL OF PARENTAL BEHAVIOR.



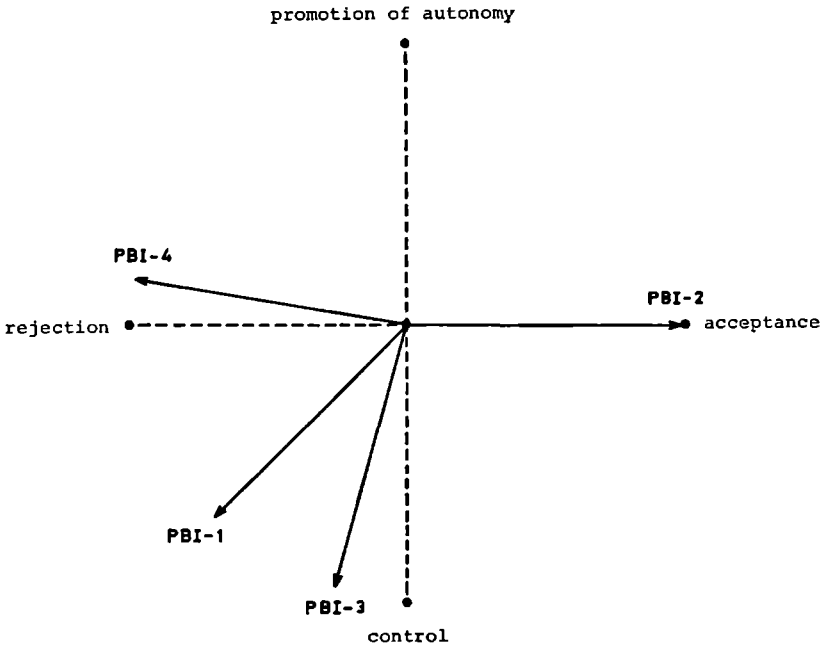
Does de Willebois, 1965). Moreover approximately 90% of alcoholics in this study are males. Deficient identification with the father's image has also been suggested as an important factor in the pathogenesis of delinquency (Mertens et al., 1965; Schouten, 1963).

Factor analysis of P.B.I. factors (and factor loadings per variable) are shown in *Appendix B.*

Fig. 4.2. shows the four P.B.I. factors in this study and their positions in Schaefer's model.

fig. 4.2.

P.B.I. - FACTORS IN THIS STUDY AND THEIR POSITIONS IN SCHAEFER'S MODEL.



ANALYTICAL EPIDEMIOLOGY: PREDISPOSING AND PERPETUATING FACTORS IN ALCOHOLISM AND ITS PRECURSOR STAGES. RESULTS.

5.1. METHODS

The predisposing and perpetuating factors were evaluated with regard to their prevalences in the diagnostic categories in the combined population of survey B (99 "controls") and survey C (125 "pathological drinkers"). This combined survey B & C population consisted of 28 abstainers, 41 social drinkers, 24 problem drinkers, 5 gamma-prealcoholics, and 126 alcoholics. Because of the small number of gamma-prealcoholics this category was incorporated into the category of alcoholics.

Evaluation of the prevalences of the predisposing and perpetuating factors was performed:

1) for male respondents younger than 30 years and for males older than 30 years separately; this was done, because - as shown in chapter 3 - alcoholics are concentrated above the age of 30, and abstainers and social drinkers below that age;
2) for males and females separately, because males prevail among alcoholics, (gamma-)prealcoholics and problem drinkers, while females prevail among abstainers.

In order to retain sufficient respondents per diagnostic category it was decided:

1) To incorporate the category of "abstainers" into the category of "social drinkers" - thus representing a category "without pathological drinking";
2) To incorporate in the group of female respondents the category of "problem drinkers" into the category of "alcoholics and gamma-prealcoholics".

Table 5.1. shows the subdivisions made for the evaluation of the predisposing and perpetuating factors.

Some of these factors were also evaluated in survey A (N = 708).

Table 5.1

Subdivisions for the evaluation of the predisposing and perpetuating factors in survey B & C (N = 224)

Subdivisions	Diagnostic categories
1) Females	a) Abstainers & Social Drinkers b) Problem Drinkers, Gamma prealcoholics & Alcoholics (i.e. Pathological Drinkers)
2) Males younger than 30 years	a) Abstainers & Social Drinkers b) Problem Drinkers c) Alcoholics & Gamma-prealcoholics
3) Males older than 30 years	a) Abstainers & Social Drinkers b) Problem Drinkers c) Alcoholics & Gamma-prealcoholics
4) All respondents	a) Abstainers & Social Drinkers b) Problem Drinkers c) Alcoholics & Gamma-prealcoholics

In survey A a fourfold subdivision was used for all respondents, i.e.:

a) Abstainers (N = 181);
b) Social Drinkers (N = 371);
c) Problem Drinkers (N = 83);
d) Alcoholics, Prealcoholics & Gamma-prealcoholics (N = 73).

Appendix C shows the methods employed to evaluate the *predisposing factors.*

Appendix D shows the methods employed to evaluate the *perpetuating factors.*

Factor analysis was performed in:

a) the combined population of survey B and survey C;
b) the population of survey A.

Ad a): Factor analysis in survey B & C (N = 224):

— P.B.I. factors (cf. Appendix B);
— Cultural crisis of alcohol consumption (cf. Chapter 3);
— Social normativity
— Oral fixation or regression

} these are described below.

Ad b): Factor analysis in survey A (N = 708):

— Social normativity
— Oral fixation I
— Oral fixation II

} these are described below.

5.2. RESULTS. PREVALENCES OF THE PREDISPOSING FACTORS IN THE DIAGNOSTIC CATEGORIES

The predisposing factors evaluated in this study can be categorized as shown in *table 5.2.*

Table 5.2.

Predisposing factors

I) *Parental factors*
1. Paternal inebriety in one's youth
2. Maternal inebrietv in one's youth
3. Interparental conflicts in one's youth
4. Parent-child conflicts in one's youth
5. Parental absence/broken home
6. Absence of father in one's youth
7. Absence of mother in one's youth
8. Paternal punitiveness (P.B.I.-1)
9. Paternal affection versus rejection (P.B.I.-2)
10. Paternal control-dominance (P.B.I.-3)
11. Paternal rejection-indifference (P.B.I.-4)
12. Low social status of one's father
13. Death of father in one's youth
14. Death of mother in one's youth
15. Parental divorce in one's youth
16. Illegitimacy
17. Parental consanguinity

II) *Factors in home family and extended family*
18. Familial inebriety (brothers, sisters, sons, daughters)
19. Inebriety in extended family

III) *Other predisposing factors*
20. Low educational level
21. Rural domicile
22. Autochthony
22a. "Genetic" autochthony
23. Male sex
24. Economic availability versus poverty

5.2.1. Parental factors.

While there is a great deal of theoretical material on the parental influences which are presumed to interfere with the child's normal development, and to create in him the need for alcohol, there has been little empirical evidence to support these theories.

Retrospective studies, implying testimonies of the patients themselves - like this study - are often the only basis for these theories.

Longitudinal studies, following the child from its pre-adolescent period through adulthood, were performed by Mc Cord & Mc Cord (1962) and by Robins et al. (1962).

In one or both of these studies the following factors were found to differentiate well between the pre-alcoholic children and those who did not become alcoholics:

— interparental conflicts;
— maternal deviance (sex, crime, inebriety);
— father's low esteem of the wife;
— mother's resentment of her role manifested in "martyrdom" and outright neglect;
— father's evasion of his role as a male;
— failure of the parents to set a clear-cut responsible male role expectation for the son;
— father's low social status;
— parental inadequacy (e.g. failure to provide physical care, financial support, supervision, or a good example);
— father's antisocial behavior (e.g. cruelty, desertion, non-support, inebriety, aggressiveness, imprisonment);
— antisocial behavior in youth on the part of the children themselves (e.g. juvenile court appearances).

The kind of pathology in early childhood related to alcoholism was described as *antisocial* rather than neurotic behavior by Robins et al., though acknowledging that their sample was not representative since it was derived from children who attended childhood clinics because of antisocial behavior in their homes (Robins et al., 1962). The remainig parental factors as mentioned in *table 5.2.* are derived from retrospective studies.

Parental rejection, parental overprotection and *parental loss,* appeared to be the most significant factors in the pathogenesis of alcoholism in Wahl's study (Wahl, 1956). No single factor is more important in the development of the child than the specific attitudes manifested towards him in early youth by significant socializers, particularly his parents. Through his parents, by the process of identification, the basic security and adequacy patterns which persist throughout life are evolved and consolidated. The developing child is exposed to fear- and guilt-inducing situations, and his defense against these takes the form of identification with strong, affectionate

parents whose high regard of him is internalized and transformed, through identification, into high self-regard, self-esteem and a self-confident approach to later problems. Without this appropriate development such a child may later recur to effective fear reducers such as alcohol and drugs (Wahl, 1956). The tendency to react in this fashion (recurrence to external fear-reducers) to stress, is internalized during childhood along with persistent feelings of unworthiness and low self-esteem; these feelings, though consciously forgotten, may later be accentuated by a subsequent stress even of a mild and transitory character (Wahl, 1956).

5.2.1.1. Paternal inebriety in one's youth.
5.2.1.2. Maternal inebriety in one's youth.

The prevalences of "*paternal inebriety*" (variable no. 99) and "*maternal inebriety*" (variable no. 100) in survey B & C are shown in *tables 5.7 & 5.8.* and *fig. 5.2.-5.5.*

The numbers applicable for "*maternal inebriety*" were too small to permit statistical analysis.

The findings concerning "*paternal inebriety*" are in agreement with previous reports (Turfboer, 1957; Wever, 1971, 1975) and with those mentioned by others (Åmark, 1951; Cork, 1969; Edwards, 1970; Fox, 1968; Robins et al., 1962; Van der Does de Willebois, 1965; Wanberg & Knapp, 1970), and are unequivocal among all subdivisions in survey B & C as shown in *tables 5.7. & 5.8.* "*Paternal inebriety*" appears to be a significant predisposing factor.

These two factors can be interpreted etiologically in the following terms

1) As a *possibly genetic factor*
   The findings (cf *table 5.7*, *table 5.8*, and *fig. 5.2.-5.5.*) are *suggestive* for genetic factors, though by no means conclusively proving. Still, by these findings the possibility of a genetic factor cannot be rejected. This possibly genetic factor may imply
   a) A postulated *Amerindian genetic factor* (Bijl, 1971, Caravedo, 1957)
   b) *Inbreeding and genetic drift* in the autochthonous Aruban population (Wever, 1971, 1975)
2) As an *acquired psychological factor* in the sense of emotional deprivation by one's parents in early youth (Cork, 1969, Edwards, 1970, Milt, 1969, Robins et al, 1962)
3) As a *sociocultural factor* in the sense of early environmental influences conducive to the *"alcoholismic learning process"* or fitting into the *"reinforcement theory of learning"* (Jellinek, 1960. Kalant & Kalant, 1971, Keller, 1972) This implies that, notwithstanding the considerable deprivation caused by parental alcoholism, the children of alcoholics may have learnt from their parents' example of inebriety, how to get instant relief for life's stresses through alcohol

Parental inebriety frequently implies

1) serious derangement of healthy personality development in the children and of healthy development of the family unit as a whole Children of alcoholics therefore are a particularly high-risk group for developing alcoholism (Cork, 1969, Fine et al , 1976, Jackson, 1962)
2) Interparental disharmony and biparental rejection of the children, perceived by the latter as the most excruciating features of parental deprivation, even as more unbearable than the alcoholic's excessive drinking *per se* (Cork, 1969, Mc Cord & Mc Cord, 1962)
3) Alcoholic parents frequently are impulsive, egocentric, irresponsible, suspicious, rigid and unable to face the realities of daily life - hence providing deficient identification possibilities for their children (Jackson, 1962, Krimmel, 1973, Wahl, 1956)
4) Deficient identification possibilities with the drinking father because of his inadequate adult male model (Cork, 1969, Mc Cord & Mc Cord, 1962, Van der Does de Willebois, 1965) This is true for sons and daughters, but especially for sons, since they may identify masculine independence with inebriety, and thus will come into conflict with what the community at large views as masculine or generally adult independence (Jackson, 1962)
5) Interparental disharmony and conflicts, veiled or open, frequently leading to fights in which for instance wives are hit by their intoxicated husbands (Cork, 1969, Mc Cord & Mc Cord, 1962, Wahl, 1956)
6) Lack of discipline, since the drinking father is not able to impose discipline on his children, but tends to play a passive role in the education of his children (Cork, 1969, Mc Cord & Mc Cord, 1962)
7) Deficient self-esteem and hence inability to face problems later in life by the alcoholic's children (Cork, 1969, Wahl, 1956)
8) Lack of easy, relaxed relationships between the alcoholic and his or her children (Cork, 1969)
9) Loss of respect by the alcoholic's children for both the alcoholic and the non-alcoholic parent (Cork, 1969)
10) Aggressive behavior by the alcoholic parent, resulting in frequent hitting of the children, fights in bars or elsewhere with friends or strangers, or the destruction of household goods (Cork, 1969, Mc Cord & Mc Cord, 1962, Wahl, 1956)
11) Outbursts of emotional lability by the alcoholic parent, expressing his notion of being unloved by his (or her) marital partner and children or his/her perception of self as being an inadequate parent, with consequent strain for the alcoholic's children (Cork, 1969)
12) Repetitive conflicts between parents and children and between the children themselves (Cork, 1969)
13) Continuous deprivation and insecurity in the alcoholic's children, in the sense of their developing anxiety, frustration, resentment, confusion, anger, socially aggressive behavior, unethical behavior, poor emotional control, schizoid withdrawal, domineering sadistic behavior, hyperactive-expansive behavior, inability to maintain attention, impulsiveness, inadequate needs for independence, inability to resist delay of sensual gratification, unresponsiveness to environmental stimulation and proneness to becoming emotionally upset or depressive (Cork, 1969, Fine et al , 1976)
14) Alienation from human relations in general in the alcoholic's children, based on the violation of confidence they experience from their alcoholic and non-alcoholic parents (Cork, 1969, Fine et al , 1976)
15) Emotional deprivation of the alcoholic's children by their non-alcoholic mothers as a reaction to husband s drinking (Cork, 1969)
16) Lack of joy in the alcoholic's family (Cork, 1969)
17) Loss of friends, i e social isolation (ostracism) of the alcoholic and his family because of the stigma attached to alcoholism by the community

(Cork, 1969, Jackson, 1962, Whalen, 1953, Yudin et al, 1976) Consequently, this implies *no* or *poor* participation in clubs, hobbies, sports, theatre, dances, etc

18) Temporary or definite separation or divorce, with consequently loss of the possibility of paternal identification examples (Cork, 1969)
19) Authority conflicts in the alcoholic's children, especially when there has been unwise use of authority by the (non-)alcoholic parent(s) (Cork, 1969)
20) Deterioration of school achievements of the alcoholic's children because of the continuous strain they live under (Cork, 1969, Whalen, 1953)
21) Damage to the physical health of the alcoholic's children, the following examples were given. chronic asthma, exogenous obesity, enuresis nocturna at age 15, chronic stomach-ache, and peptic ulcer (Cork, 1969)
22) Traffic and other accidents or imprisonment of the alcoholic because of traffic violations or other violations of the law, with subsequent increased strain in the alcoholic's children (Cork, 1969)
23) Lax or erratically punitive disciplinary methods of alcoholic fathers towards their children These two disciplinary methods are also significantly involved in the causation of certain types of criminal behavior (Mc Cord & Mc Cord, 1962)
24) Feelings in the alcoholic parent of being victimized by society, compensatory feelings of grandiosity, a tendency to place enjoyment above other values (hedonism), and a tendency to openly express dependency needs (Mc Cord & Mc Cord, 1962)

5.2.1.3. Interparental conflicts in one's youth

The prevalences of *interparental conflicts* (variable no. 124) are shown in *tables 5.7. & 5.8.* and *fig. 5.3.-5.5.* Responses other than "harmonious, pleasant" or "not applicable" were considered as indicative of interparental conflicts.

This factor (cf table 4 3) can be interpreted etiologically as an *early sociocultural factor in the microclimate* (Edwards, 1970, Jellinek, 1945, Jones, 1966, Mc Cord & Mc Cord, 1962, Robins et al, 1962, Van der Does de Willebois, (1965) This factor measures the dimension of emotional deprivation in childhood, generated by frustrating unstable interparental relationships, briefly relationships which could have been improved through cooperation of those involved This in contrast to a factor such as death of father, which to a great extent measures childhood deprivation generated by *fate*

Lack of family integration in childhood was previously found as a conditioning factor in the development of alcoholism (Cork, 1969, Jones, 1966, Robins et al, 1962, Van der Does de Willebois 1965), as well as general *parental inadequacy*, and especially *paternal antisocial behavior* even in the absence of paternal inebriety (Cork, 1969, Robins et al, 1962) Intense interparental conflicts in childhood may lead to alcoholism in later life (Mc Cord & Mc Cord, 1962)

The findings in this study are in agreement with previous reports (Wever, 1971, 1975) and with those mentioned above, though *not* statistically significant among female respondents. This may indicate that only *boys* are stimulated on the alcoholismic gradient by this factor.

However, for males older than 30 years, and for the total of all respondents the correlation is less convincing (cf. table 5.8.).

5.2.1.4.Parent-child conflicts in one's youth

The prevalences of "*parent-child conflicts*" (variable no. 134) are shown in *tables 5.7. & 5.8.* Responses other than "harmonious, pleasant" or "not applicable" were considered as indicative of parent-child conflicts. This factor can be interpreted etiologically in the same terms as the factor *interparental conflicts in youth.*

The findings, as shown in *tables 5.7. & 5.8.* and *fig. 5.3.*, reveal statistically significant differences between the diagnostic categories *only for male respondents younger than 30 years:* only in this group this dimension ("parent-child conflicts") seems to be a significant predisposing factor.

5.2.1.5. Parental absence (broken home) in one's youth

The prevalences of this factor are shown in *tables 5.7. & 5.8.* Appendix C shows the method used to evaluate this factor. This factor can be interpreted etiologically in the same terms as the two preceding factors.

The findings shown in *tables 5.7. & 5.8.* reveal *no* statistically significant differences between the diagnostic categories, and hence are at *variance* with those of Cahalan et al., i.e. correlation between "broken homes in early youth" and heavy drinking in males (Cahalan et al., 1968, 1969), as well as with the views concerning "lack of family integration", "parental inadequacy" and "parental antisocial behavior" as predisposing factors (Jones, 1966; Robins et al., 1962; Van der Does de Willebois, 1965; Wanberg & Knapp, 1970), as well as with Wahl's observation of "loss of parent(s) by death or separation before age 15" in 37% of 109 male alcoholics (Wahl, 1956).

5.2.1.6 Absence of father in one's youth

5.2.1.7. Absence of mother in one's youth

The prevalences of "*absence of father*" (variable no. 97) and "*absence of mother*" (variable no. 98) are shown in *tables 5.7. & 5.8.*

Both these factors can be interpreted etiologically in the same terms as the three preceding factors. The findings reveal *no* statistically significant differences between the diagnostic categories.

5.2.1.8. Paternal punitiveness (P.B.I.-1)

5.2.1.9. Paternal affection versus rejection (P.B.I.-2)

5.2.1.10. Paternal control-dominance (P.B.I.-3)

5.2.1.11. Paternal rejection-indifference (P.B.I.-4)

The prevalences of these four P.B.I.-factors are shown in *table 5 9.* and *fig. 5.3.-5.4.* Appendix C shows how these four factors were evaluated.

The percentages shown in *table 5.9.* and *figs. 5.3 -5.4.* represent the percentages of *below median* factor scores for P.B.I.-1, P.B.I.-3 and P.B.I.-4, and *above median* factor scores for P.B I.-2. Median factor score is for all four P.B.I.-factors: 500.

It should be emphasized that - except for P.B.I.-2 - *decreasing* factor scores were expected to correlate with the "tendency to male alcoholism" (alcoholismic gradient), since the first answers per variable in the P.B.I. questionnaire (answer no. 1) which maximally correlate with the tested variable, were given the lowest scores, cf. *appendix B.*

These four P B I -factors specifically measure dimensions of *deprivation in childhood generated by frustrating father-son-relationships* This is perfectly clear for P B I -1, P B I -3 and P B I -4, but not for P B I -2

P B I -2 measures "paternal affection" maximally at one extreme, hence "*paternal rejection*" at the other extreme Briefly, these P B I -factors measure relationships which could have been improved through cooperation of fathers involved These four factors may lead to inadequate identification and hence low self-esteem and lack of self-confidence when approaching problems later in life (Van der Does de Willebois, 1965, Wahl, 1956)

Such an identification crisis may provoke recurrence to effective external fear reducers such as drugs and alcohol (Wahl, 1956)

Paternal punitiveness (P B I -1) was indicated as a predisposing factor to alcoholism (Cork, 1969, Criteria Committee, 1972, Edwards, 1970, Robins et al 1962, Wahl, 1956)

The other three P B I -factors are fairly close to P B I -1, as shown in *fig 4 2*

Paternal rejection(-indifference), as reflected by P B I -4 and P B I -2, was also indicated as a predisposing factor (Cork, 1969, Criteria Committee, 1972, Edwards, 1970, Mc Cord & Mc Cord, 1962, Wahl, 1956), and was found far more frequently than *maternal* rejection (Wahl, 1956) The combination of paternal alcoholism and paternal rejection was found to be productive of alcohol addiction in sons of such fathers (Mc Cord & Mc Cord, 1962)

The results are shown in *table 5 9.* and *figs 5.3.-5 4.*, and reveal the following characteristics:

a) *P B.I.-1: No* significant correlation for male respondents younger than 30 years. Significant correlations for male respondents older than 30 years *and* for male respondents of all ages. However, for

the latter two groups of respondents these significant correlations were *rejected* since they were difficult to interpret: problem drinkers appeared to have a *lower* percentage than abstainers plus social drinkers *or* alcoholics.

b) *P.B.I.-2:* A significant correlation was only found for male respondents younger than 30 years. Hence in this age group *paternal rejection* is a significant predisposing factor.

c) *P.B.I.-3:* A significant correlation was found only for male respondents older than 30 years *and* for male respondents of all ages. Hence for this age group (>30) *paternal control-dominance* is a significant predisposing factor.

d) *P.B.I.-4:* A significant correlation was only found for male respondents of all ages. Therefore *paternal rejection-indifference* is considered a significant predisposing factor for males younger *and* older than 30 years.

No explanation could be found for the observed differences of P.B.I.-2 (paternal rejection), P.B.I.-3 (paternal control-dominance), and P.B.I.-4 (paternal rejection-indifference) as predisposing factors among either males younger than 30 years or older than 30 years; the influence of age thus observed is not understood.

5.2.1.12. Low social status of one's father

The prevalences of this factor (variable no. K) are shown in *tables 5.7. & 5.8.*

The type of social stratification used in this study has been derived from the G.T.E. (Nijmeegse werkgroep voor Gezinstypologie en Epidemiologie) in use at the Department of Family Medicine at Nijmegen University, and was adapted to Aruban circumstances.

Appendix E shows a classification of the professions observed in this study and the adapted scores assigned to them. Scores are identical to the numbering of the social classes. High scores correlate with low social class, and low scores with high social class.

The findings reveal *no* significant correlations, except for the group of all respondents, and for male respondents older than 30 years. These significant correlations were *rejected,* however, because of interpretation difficulties: problem drinkers appeared to have a *lower* percentage than abstainers plus social drinkers *or* alcoholics. It can be speculated that this factor - like P.B.I.-1 among males older

than 30 years - could work in both directions, i.e. as a predisposing factor to both alcoholism and abstinence.

The results are at variance with reports indicating a correlation between the development of alcoholism and low social status of fathers of future alcoholics (Cahalan & Cisin, 1968; Cahalan et al., 1969; Robins et al., 1962).

The construction of a social stratification system is a source of problems for most investigators (De Vries, 1973). The purpose of such a classification is twofold:

a) an attempt to describe a socio-economic reality;
b) an attempt to achieve comparability between the population studied and other populations which were already stratified in a certain manner; this often leads to rigid stratification systems which in the long run distort reality.

In choosing a stratification method the following factors play an important role:

1) the purposes of the investigator;
2) the character of the criteria, chosen by the investigator;
3) the number of criteria to be employed;
4) the character of the population to be studied;
5) other populations, with which comparisons are made;
6) the reliability of the measuring instrument;
7) a filling of the social classes, acceptable for statistical processing.

The criteria of profession, professional autonomy, educational level and social prestige are those most often applied, and in this category generally profession plays the most important role. The ***G.T.E.*** as employed in this study, operates with criteria like profession, educational level and professional independence (De Vries, 1973).

5.2.1.13. Death of father in one's youth

5.2.1.14. Death of mother in one's youth

The prevalences of these factors (variable no. 95 and 96 resp.) are shown in *tables 5.7. & 5.8.*

The results reveal *no* statistically significant correlations, and hence are at variance with Wahl's statement that loss of the father by death (or separation) operates as a major and enduring stress situation especially among boys (Wahl, 1956). The somewhat higher prevalence of early loss of father by death (21% of alcoholics and gamma-prealcoholics) as compared to early loss of mother by death (13% of alcoholics and gamma-prealcoholics) is, however, in agreement with Wahl's observations, and suggests that the relationship to the *father* plays a crucial role in the development of the *son* (Wahl, 1956). Both these factors can be interpreted etiologically in the same terms as the factors: parental absence; absence of father; absence of mother; parental divorce; and illegitimacy.

5.2.1.15. Parental divorce in one's youth

The prevalences of this factor (variable no. 94) are shown in *tables 5.7. & 5.8.*

The results indicate, that there is *no* correlation between parental divorce in early youth and the shift on the alcoholismic gradient. "Loss of parents by separation or divorce" was found earlier in 10% of 109 male alcoholics (Wahl, 1956).

5.2.1.16. Illegitimacy

The prevalences of this factor (variable no. 119a) are shown in *tables 5.7. & 5.8.*

This factor can be interpreted etiologically in the same terms as the factors *parental divorce, parental absence in youth* and *death of father in youth.* This dimension (*illegitimacy*) may be caused by parents' choice or by fate (e.g. the case of a common-law relationship between a married man and an unmarried woman). In most instances it implies prolonged absence of the biological father in the family unit of an unmarried woman with her illigitimate children.

The results in *tables 5.7. & 5.8.* indicate however, that illegitimacy generally does *not* play an important role in the etiology of alcoholism in Aruba. The so-called "Caribbean family type" (Beaubrun, 1971; Hoetink, 1957) consisting of an unmarried woman with her illegitimate children from one or more fathers - often occurring among the Negroid population of the West-Indies - has had no apparent influence in the development of alcoholism; this is in agreement with Beaubrun's findings (1971), and at variance with Edward's statement (1970), that illegitimacy plays a role in the etiology of alcoholism. Two other variables indicative for the factor *illegitimacy* (variables no. 118 and 119) were omitted, because they measure this dimension in synonymous ways.

5.2.1.17. Parental consanguinity

The prevalences of this factor (variable no. 120) are shown in *tables 5.7. & 5.8.*

This factor may imply a *possibly genetic factor: inbreeding* in the original autochthonous population (Wever, 1971, 1975) as well as

in the remaining population of Aruba, possibly resulting in higher prevalences of alcoholism and its precursor stages in consanguineous marriages.

Tables 5.7. & 5.8. give *no* support for this hypothesis.

5.2.2. Predisposing factors in home family and extended family

No single factor is more important in the development of the child than the specific attitudes manifested towards him in early life by significant socializers, particularly his parents (Wahl, 1956). In a culture, in which the extended family plays an important role in the education of the child - as happens to be the case in Aruba like in most Latin American countries - it is conceivable that apart from parents, other relatives (siblings, grandparents, aunts, uncles, cousins) may have some influence in the development of alcoholism, though of course their influence will be less important than the specific parental influences. In this category only two factors were evaluated: *familial inebriety* and *inebriety in extended family.*

5.2.2.1. Familial inebriety

5.2.2.2. Inebriety in extended family

The prevalences of both these factors (variables no. 104 and no. 105. resp.) in survey B & C are shown in *tables 5.7. & 5.8.* and *figs. 5.2.-5.5.*

The prevalences of *familial inebriety* in survey A are shown in *table 5.10.*

The findings concerning *familial inebriety* in survey B & C reveal *no* statistically significant correlations among female respondents or among male respondents younger than 30 years. Among males older than 30 years and in the group of all respondents there *are* significant correlations. However, both these latter significant correlations were *rejected,* because of the same type of interpretation difficulties as described previously sub *5.2.1.13.* and *5.2.1.8.* In survey A, however, there is significant statistical correlation for this factor.

The findings concerning *inebriety in extended family* reveal significant correlations among *all* subdivisions evaluated.

The results concerning *familial inebriety* (in *survey A)* and *ine-*

briety in extended family (in *survey B & C)* are in agreement with previous reports (Wever, 1971, 1975) and with statements by other authors (Åmark, 1971; Edwards, 1970; Fox, 1968; Mac Clearn, 1973; Wanberg & Knapp, 1970).

5.2.3. Other predisposing factors

These include the following factors: low educational level; rural domicile; autochthony; male sex. The implications of these factors for the etiology of alcoholism will be dealt with under the separate sections dealing with these factors.

5.2.3.1. Low educational level

The prevalences of *(low) educational level* (variable no. 20) are shown in *tables 5.7. & 5.8.* and *figs. 5.2.-5.5.*

This factor can be interpreted etiologically in the following terms:

a) as possibly reflecting a mild degree of mental retardation (Esser, 1967): a congenital quality by which the affected individual is predisposed to alcoholism because its implications - decreased intellectual capacities - will impede him to optimally understand the dangers of excessive drinking; this implies a *congenital genuine psychiatric disease* as a predisposing *psychological factor;*
b) as an *acquired psychological factor early operative in the microclimate:* minimal development of congenital average level intellectual functions through school education, resulting in the same predisposition to alcoholism as mentioned sub a).

The findings reveal significant correlations among all subdivisions evaluated, except female respondents where statistical significance is *nearly* reached. Because the correlations are clear among all (except female) respondents, this factor is still considered as an important predisposing factor, even among females.

5.2.3.2. Rural domicile

The prevalences of *rural (-urban) domicile* (variable no. 1) in survey B & C are shown in *tables 5.7 & 5.8.* and *figs. 5.3.-5.5.*

The prevalences of *rural (-urban) domicile* in survey A are shown in *table 5.10.*

The trend in both surveys is the same: alcoholism, gamma-prealcoholism, prealcoholism, and problem drinking, are more prevalent in the *rural districts,* i.e. among all respondents and male respondents younger than 30 years, but *not* among females nor among males

older than 30 years. Therefore "rural domicile" is rejected as a predisposing factor among females. It is maintained, however, as a predisposing factor among males older than 30 years, because of the significant correlations for the subdivisions "males younger than 30 years" and "all respondents".

The findings are in agreement with previous reports concerning *rural* prevalence of alcoholism in Aruba (Berkley, 1956; Janssen, 1971; Wever, 1971, 1975), and at variance with findings in Western European and American studies where alcoholism, problem drinking or excessive drinking were found to correlate with increasing degrees of *urbanization* (Cahalan et al., 1969; Gadourek, 1963; Wallace, 1972). Gadourek views this correlation as a reflection of drinking due to decreased social participation and increased stress in urban life i.e. sociocultural deprivation in urban life (Gadourek, 1963).

Possible explanations for our contrasting findings:

a) *Interference with the factor autochthony;* autochthonous Arubans are highly concentrated in the rural districts as shown in *table 5.3.*

b) *Sociocultural deprivation in the rural districts.* It can be argued, that the rural life style during the past two centuries has implied more deprivation than the "urban" life style of Oranjestad and more recently of San Nicolás, since poverty has been concentrated in the rural districts and hence also the "culture of poverty": poor housing conditions, virtual absence of landed property, insufficient educational facilities, illegitimacy, low social status and lack of recreational facilities (Berkley, 1956, 1957). To this can be added sociocultural deprivation directed towards the original autochthonous Amerindian population by the colonizers (Dozier, 1966); the original Amerindian population has always been predominant in the rural districts (Hartog, 1953).

However, even when the factor "nationality" (variable no. 4) is held constant, the same trend is discernible, as shown in *table 5.3.*

Table 5.4. shows the prevalences of abstinence, social drinking, problem drinking, and alcoholism (& gamma-prealcoholism & prealcoholism) among urban Arubans, rural Arubans, urban Non-Arubans and rural Non-Arubans.

Table 5.3.

Prevalences of Arubans (variable no. 4) per district (variable no. 1) in percent, in survey A.

District	N	Arubans	Non-Arubans
Urban:			
San Nicolás	173	60	40
Oranjestad	204	77	23
Rural:			
Sabaneta & Brazil	104	83	17
Santa Cruz	101	93	7
Nort	75	95	5
Paradera	51	98	2

$\chi^2 = 75.68$
df = 5
p = 0.0000

Table 5.4.

Prevalences of abstinence, social drinking, problem drinking, and alcoholism (& gamma-prealcoholism & prealcoholism), according to rural-urban domicile (variable no. 1) and nationality (variable no. 4) in percent, in survey A.

Diagnostic category	Nationality and Urban-Rural Domicile			
	Urban Arubans (N = 259)	Rural Arubans (N = 301)	Urban Non-Arubans (N = 118)	Rural Non-Arubans (N = 30)
Abstinence	33	20	28	10
Social Drinking	48	52	60	70
Problem Drinking	10	16	5	10
Alcoholism (& Gamma-prealcoholism & Prealcoholism)	10	12	7	10

$\chi^2 = 28.17$
df = 9
p <0.005

5.2.3.3. Autochthony

The prevalences of this factor in survey B & C are shown in *tables 5.7. & 5.8.*

When anthropologists use the term "autochthonous population" they intend to indicate the original population of a country, region or island. In this sense, only Arubans of purely Amerindian ancestry could be considered as autochthonous. However, the definition of this concept (autochthony) becomes continu-

ously more difficult because of the increasing migration of population groups This problem implies one major question back to which origin in time does one wish to return to consider someone as autochthonous? Hence, the anthropological concept of autochthony is difficult in its application (De Vries, 1973)

From a *socio-cultural* standpoint, the term "autochthonous population" indicates those locally raised, the non-imported members of a population, those who spent the greater part of their youth within the culture of a given population (De Vries, 1973) In the latter sense the concept "authochthony" was used in this study

Appendix C shows the factor autochthony as operatized in this study.

The findings as shown in *tables 5.7. & 5.8.* reveal significant correlations *only* for *male respondents older than 30 years.*

The prevalence of variable no. 4 (*"nationality"*) - one of the three constituent variables of the factor autochthony - are shown in *table 5.10.* for survey A.

The results in survey A shown in *table 5.10.* indicate that both problem drinking and alcoholism (plus precursor stages) are more prevalent among Arubans.

This factor ("autochthony") can be interpreted etiologically in the following terms

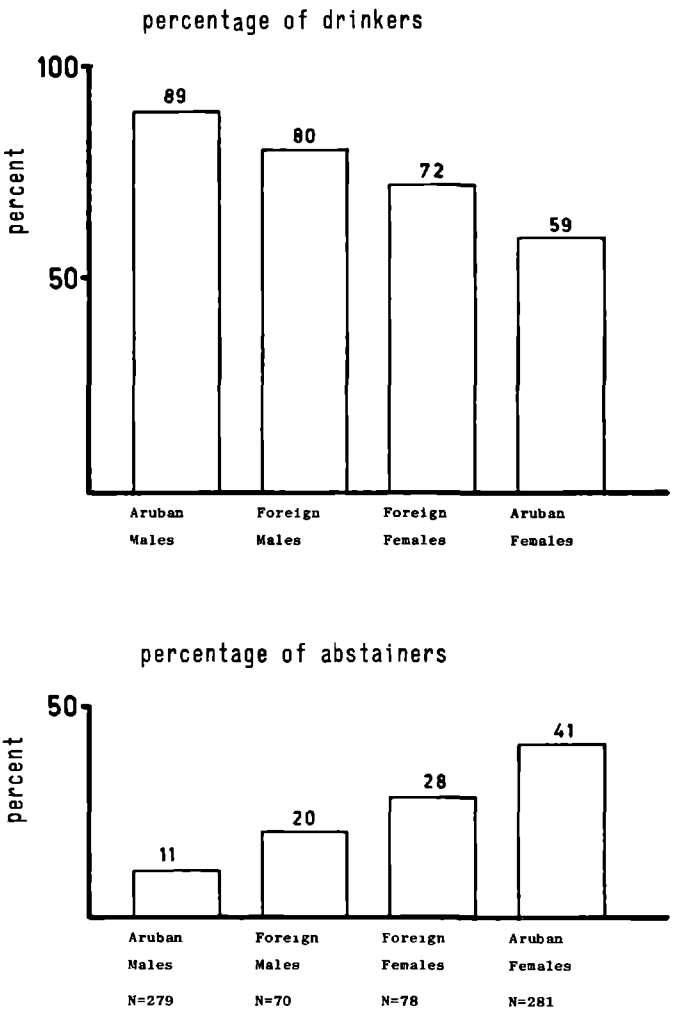
1) As a *possibly genetic factor,* implying:
   a a *postulated Amerindian genetic factor* (Bijl, 1971, Caravedo, 1957),
   b. *inbreeding* and *genetic drift* in the original autochthonous Aruban population (Wever, 1971, 1975)
2) As a *sociocultural factor* in the sense of early environmental moments conducive to the "alcoholismic learning process" (Albrecht, 1973, Bacon, 1973, Jellinek, 1960, Kalant & Kalant, 1971, Keller, 1972)

Fig. 5.1. shows the percentages of *drinkers* and *abstainers* among Aruban males, foreign males, foreign females and Aruban females.

As may be seen in *fig. 5.1.*, the prevalence of drinkers increases from Aruban females through foreign females and foreign males to Aruban males, while the prevalence of abstainers increases in the opposite direction. Hence there is greater discrepancy as regards drinking behavior between Aruban males and Aruban females, than between foreign males and foreign females, reflecting possibly a lesser degree of *emancipation* in drinking customs among Aruban females than among foreign females. This is consistent with the suggested sociocultural factor in the sense of early environmental moments conducive to the "alcoholismic learning process" (Albrecht, 1973; Bacon, 1973; Jellinek, 1960; Keller, 1972; Kalant & Kalant, 1971) and with social pressure against unrestrained female drinking

figure 5.1.

PREVALENCES OF DRINKERS AND ABSTAINERS
ACCORDING TO NATIONALITY AND SEX



(Knupfer & Room, 1964). In groups where the prevalence of drinkers and excessive drinkers is high, the prevalence of pathological drinkers will also be high (Chu, 1972). This explains *partially* the observed higher prevalence of alcoholism among Aruban males as compared with the three other groups in *table 5.6.* (Wever, 1971, 1975).

Table 5.5. shows mean scores for annual frequencies of alcohol consumption and alcohol intoxication (excessive drinking) among *drinking* Aruban males, foreign males, foreign females and Aruban females.

Table 5.5.

Mean scores for annual frequencies of alcohol consumption (variable no. 36) and alcohol intoxication (variable no. 37) according to nationality (variable no. 4) and sex (variable no. 5) in survey A among non-abstainers.

Nationality and Sex	N	Mean scores for annual frequency of alcohol consumption	Mean scores for annual frequency of alcohol intoxication
Aruban Males	248	4.1	2.9
Foreign Males	56	4.5	2.0
Foreign Females	56	4.2	1.0
Aruban Females	167	3.5	1.4

The results in *table 5.5.* are consistent with Chu's statement, since higher annual frequencies of *intoxication* among Aruban male drinkers coincide with the observed higher prevalence of alcoholism (& gamma-prealcoholism & prealcoholism) among this group. The results in *table 5.5.* also indicate, that higher prevalences of *moderate* drinking (mean annual frequency of alcohol *consumption*) do *not* necessarily imply higher prevalences of *pathological* drinking (Chu, 1972).

Table 5.6. shows the prevalences of abstinence, social drinking, problem drinking and alcoholism (& gamma-prealcoholism & prealcoholism) among Aruban males, foreign males, foreign females and Aruban females, in survey A.

The results in *table 5.6.* are consistent with the findings in *table 5.5.*

Table 5.6.

Prevalences of abstinence, social drinking, problem drinking, and alcoholism (& gamma-prealcoholism & prealcoholism), according to nationality (variable no. 4) and sex (variable no. 5) in percent, in survey A.

Nationality and Sex	N	Abstinence	Social Drinking	Problem Drinking	Alcoholism & Gamma-prealcoholism & Prealcoholism
Aruban Males	279	11	50	18	21
Foreign Males	70	20	57	10	13
Foreign Females	78	28	67	3	3
Aruban Females	281	41	50	9	1
(Total)	(708)				

$\chi^2 = 105.48$
$df = 9$
$p < 0.005$

5.2.3.3.1. "Genetic" autochthony

The prevalences of this factor are shown in *tables 5.7. & 5.8.* and *figs. 5.4-5.5.*

Appendix C shows this factor as operatized in this study.

The findings reveal significant correlations only for the subdivisions "all respondents" and "male respondents older than 30 years". Hence it seems evident that this factor - as well as the preceding one, i.e. autochthony (in a sociocultural sense) - is only an important predisposing factor among males older than 30 years, but *not* among females nor among males younger than 30 years. This is in agreement with a previous observation that alcoholism prevalence in Aruba is *increasing* among non-Aruban males as compared with the nineteen-fifties, probably due to acculturation of non-Arubans to Aruban drinking customs (Wever, 1971, 1975). This latter remark, of course, is a strong support for the view that alcoholism would be a learned behavior (Albrecht, 1973; Bacon, 1973; Cahalan, 1976; Jellinek, 1960; Kalant & Kalant, 1971; Keller, 1972).

This factor ("*genetic*" *autochthony*) can be interpreted etiologically as a *possibly genetic factor*, as previously described sub 4.4.3.3.

5.2.3.4. Male sex

Male sex prevalences (variable no. 5) in survey B & C and in survey A are shown in *tables 5.8. & 5.10.* and *fig. 5.5.*

The results, showing a high correlation between male sex and the alcoholismic gradient, are in agreement with other studies (Cahalan

& Cisin, 1968; Cahalan et al., 1969; Esser, 1965; Gadourek, 1963; Knupfer & Room, 1964; Wilkinson et al., 1969) and with previous findings in Aruba (Oldenboom, 1971; Wever, 1971, 1975).

This factor can be considered to have a number of etiological interpretations

1) A sociocultural factor in the sense of early environmental moments conducive to the development of the *virility complex* the (excessive) use of alcohol to symbolize manhood and adult life in males (Gadourek, 1963, Hoetink, 1957)

   This is congruent with Gadourek's *role theory* (excessive) drinking as essential token of society's expectation of the *adult* role especially for *males*, less for females (Gadourek, 1963)

   It was stated previously that in Aruba alcohol consumption in males is stimulated to symbolize manhood, and that an abstinent youngster may not be considered as a "real" man but as a "homosexual," and also that men may use alcohol to minimalize their inhibitions when intending to have extramarital sexual contacts, which behavior *per se* is considered as a token of virility (Wever, 1971)

2) A sociocultural factor in the sense of universally prevailing *social pressure against unrestrained drinking by women* (Knupfer & Room, 1964) This social pressure can be viewed as

   a) A reflection of women's economic dependence on men, i e women generally do not earn their living by paid work, and hence depend on men financially which implies that women are not expected to freely dispose of men's financial support e g in the sense of excessive expenditures on alcoholic beverages,

   b) A reflection of the value placed on sexual purity in women, defined as conjugal fidelity and absence of extramarital or premarital promiscuity, release of inhibitions due to drinking might lead women to violate approved standards of sexual restraint (Knupfer & Room, 1964)

   It is evident that for *men*, contrary to women, there exists social pressure against *abstinence*, since men are generally financially independent, and because among men conjugal infidelity and extramarital or premarital promiscuity, at least in the Latin American setting, are considered as tokens of virility

3) A reflection of possibly *genetic factors* in the sense of (male) *sex limitation* or *X-linkage* (Mc Clearn, 1973)

5.2.3.5. Poverty versus economic availability of alcohol

The prevalences of this factor are shown in *tables 5.7. & 5.8.* and *figs 5.3.-5.5.*

Appendix C shows the method used to evaluate this factor.

The findings reveal *no* significant correlations except for the subdivisions "male respondents younger than 30 years" and "all respondents". These significant correlations are, however, *opposite* to the original expectation ($R = -0.43388$, $R = -0.13098$ resp.), which includes that the alcoholismic gradient - at least among the subdivisions mentioned - correlates with *economic availability* and not with poverty.

Theoretically, this factor can be interpreted as follows

a) *Poverty* can be caused by alcoholism, but reciprocally alcoholism can be

perpetuated, provoked and stimulated by poverty, in this sense this factor reflects *"escapism', 'escape drinking', 'excessive drinking to escape from one's stressful reality"* (Bell et al., 1976, Cahalan et al., 1969, Edwards, 1970, Esser, 1965, Gadourek, 1963, Jellinek, 1960, Kalant & Kalant, 1971) and the *"culture of poverty'* as described in the section "rural domicile", *a late sociocultural factor in the microclimate*

b) *Economic availability* can be viewed as both a *late sociocultural factor in the microclimate* and as a *sociocultural factor in the macroclimate* in the sense of *economic availability* of alcohol (the relatively low cost of alcoholic beverages as compared to a population's modal or an individual's per capita income) (De Lint & Schmidt, 1970, 1971, 1971a, 1975, Esser, 1965, Gadourek, 1963, Wallace, 1972) This does *not* represent a circular causality, however, since alcoholism does not perpetuate nor stimulate wealth

The findings are in agreement with the latter view (sub b) and with observations by others (Cahalan & Cisin, 1968; Cahalan et al., 1969, Gadourek, 1963; Wallace, 1972).

Table 5 7

Predisposing factors in percent per diagnostic category for female respondents

Factor	Diagnostic category		χ^2	df	p
	Abstinence & Social Drinking (N = 24)	Problem Drinking & Alcoholism & Gamma-prealcoholism (N = 14)			
Paternal inebriety in one's youth	17	64	7 98	1	<0 005
Maternal inebriety in one's youth	0	7	—	—	—
Interparental conflicts in one's youth	33	43	0 49	1	n s
Parent-child conflicts in one's youth	25	29	—	—	—
Parental absence/broken home	29	36	—	—	—
Absence of father in one's youth	25	21	—	—	—
Absence of mother in one's youth	8	14	—	—	—
Low social status of one's father	50	50	0 00	1	n s
Death of father in one's youth	21	7	—	—	—
Death of mother in one's youth	13	7	—	—	—
Parental divorce in one's youth	4	14	—	—	—
Illegitimacy	17	29	—	—	—
Parental consanguinity	0	7	—	—	—
Familial inebriety	25	7	—	—	—
Inebriety in extended family	25	57	4 40	1	<0 05
Low educational level	17	43	3 08	1	n s
Rural domicile	42	36	0 30	1	n s
Autochthony	92	71	3 53	1	n s
'Genetic autochthony	50	36	0 47	1	n s.
Poverty vs economic availability	71	79	0 58	1	n s

χ^2 was not applied for those factors where the figures were too low for statisical analysis

Table 5 8

Predisposing factors in percent per diagnostic category
a) male respondents younger than 30 years (= M < 30)
b) male respondents older than 30 years (= M > 30)
c) all female and male respondents (= F + M)

Factor	Diagnostic category			R	p
	Abstinence & Social Drinking	Problem Drinking	Alcoholism & Gamma-prealcoholism		
Paternal inebriety in one's youth					
M <30	8	56	40	0 35744	0 0043
M >30	0	46	41	0 18865	0 0031
F + M	9	46	44	0 30150	0 0000
Maternal inebriety in one's youth					
M <30	0	0	0	—	—
M >30	0	0	1	—	—
F + M	0	0	2	—	—
Interparental conflicts in one's youth					
M <30	16	56	60	0 41942	0 0024
M >30	20	0	34	0 14541	0 0127
F + M	23	21	37	0 14086	0 0125
Parent-child conflicts in one's youth					
M <30	4	33	40	0 33471	0 0029
M >30	20	8	18	0 01646	n s
F + M	16	17	21	0 05190	n s
Parental absence/broken home					
M <30	12	11	30	0 12397	n s
M >30	25	23	27	0 01587	n s
F + M	22	17	28	0 06920	n s
Absence of father in one's youth					
M <30	20	22	20	0 00620	n s
M >30	35	23	38	0 04602	n s
F + M	26	21	35	0 09391*	0 0669
Absence of mother in one's youth					
M <30	8	0	30	0 13223	n s
M >30	15	15	14	-0 00972	n s
F + M	10	8	15	0 05190	n s
Low social status of one's father					
M <30	40	56	50	0 11364	n s
M >30	55	39	66	0 11684	0 0467
F + M	48	46	63	0 15322	0 0113
Death of father in one's youth					
M <30	8	11	0	-0 04752	n s
M >30	15	23	24	0 04463	n s
F + M	15	17	21	0 05684	n s
Death of mother in one's youth					
M <30	0	0	10	0 07025	n s
M >30	15	15	14	0 00972	n s
F + M	9	8	13	0 04201	n s

Parental divorce in one's youth					
M <30	12	11	10	-0.01653	n.s.
M >30	5	8	6	-0.00258	n.s.
F + M	7	8	7	-0.00494	n.s.
Illegitimacy					
M <30	8	11	10	0.02273	n.s.
M >30	15	8	13	0.00139	n.s.
F + M	13	8	15	0.01977	n.s.
Parental consanguinity					
M <30	4	0	0	-0.03926	n.s.
M >30	0	0	8	0.05892*	0.0459
F + M	1	0	8	0.06178*	0.0175
Familial inebriety					
M <30	4	11	10	0.06198	n.s.
M >30	15	8	29	0.11922	0.0281
F + M	15	13	25	0.10627	0.0262
Inebriety in extended family					
M <30	8	22	40	0.26446	0.0146
M >30	10	23	47	0.23249	0.0005
F + M	15	25	47	0.30644	0.0000
Low educational level					
M <30	4	11	40	0.27273	0.0057
M >30	20	23	39	0.12517	0.0332
F + M	13	21	39	0.24219	0.0000
Rural domicile					
M <30	36	33	70	0.23140	0.0686
M >30	35	69	57	0.07756	n.s.
F + M	38	54	56	0.15569	0.0109
Autochthony					
M <30	84	100	90	0.08678	n.s.
M >30	60	85	85	0.12418	0.0126
F + M	75	88	85	0.07661	n.s.
„Genetic" autochthony					
M <30	56	56	60	0.02686	n.s.
M >30	40	54	74	0.21305	0.0008
F + M	49	50	70	0.20265	0.0011
Male sex					
F + M	65	92	91	0.23007	0.0000
Poverty versus economic availability					
M <30	64	44	10	-0.43388	0.0029
M >30	35	23	41	0.07221	n.s.
F + M	58	38	42	-0.13098	0.0264

R = Kendall's Tau C. * R not significant if: —0.10 <R< +0.10.

Kendall's Tau C was not applied for those factors where the figures were too low for statistical analysis.

M <30 : *25* abstainers & social drinkers, *9* problem drinkers, *10* alcoholics & gamma-prealcoholics.

M >30 : *20* abstainers & social drinkers, *13* problem drinkers, *109* alcoholics & gamma-prealcoholics.

F + M : *69* abstainers & social drinkers, *24* problem drinkers, *131* alcoholics & gamma-prealcoholics.

Table 5 9

P B I -factors in percent per diagnostic category
a) male respondents younger than 30 years (= M < 30)
b) male respondents older than 30 years (= M > 30)
c) all male respondents (= M)

Factor	Diagnostic category			R	p
	Abstinence & Social Drinking	Problem Drinking	Alcoholism & Gamma-prealcoholism		
Paternal punitiveness (= P B I -1)					
M <30	46	43	75	0 11387	n s
M >30	35	25	58	0 21093	0 0181
M	41	33	59	0 19046	0 0232
Paternal affection vs rejection (= P B I -2)					
M <30	41	71	100	0 39302	0 0101
M >30	41	63	47	0 00498	n s
M	41	67	50	0 06148	n s
Paternal control-dominance (= P B I -3)					
M <30	18	14	25	0 01102	n s
M >30	41	50	70	0 22256	0 0113
M	28	33	67	0 38150	0 0000
Paternal rejection-indifference (= P B I -4)					
M <30	32	57	50	0 19467	n s
M >30	47	50	58	0 08803	n s
M	39	53	58	0 17610	0 0327

R = Kendall's Tau C

M <30 *22* abstainers & social drinkers, *7* problem drinkers, *4* alcoholics & gamma-prealcoholics

M >30 · *17* abstainers & social drinkers, *8* problem drinkers, *60* alcoholics & gamma-prealcoholics

M *39* abstainers & social drinkers, *15* problem drinkers, *64* alcoholics & gamma-prealcoholics

figure 5.2

Females predisposing factors.

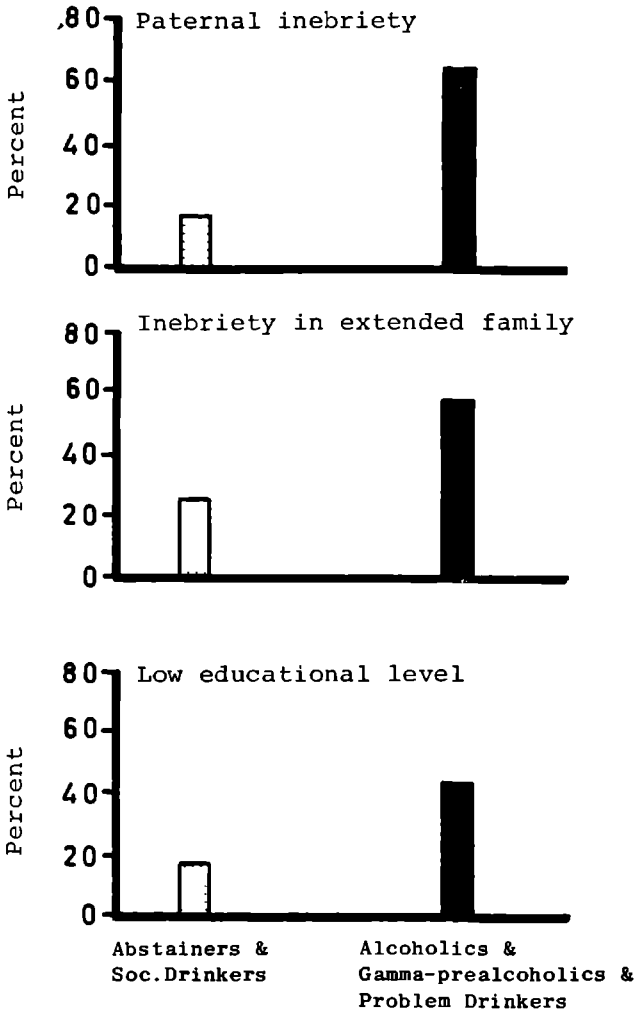


figure 5 3

Males younger than 30 years predisposing factors.

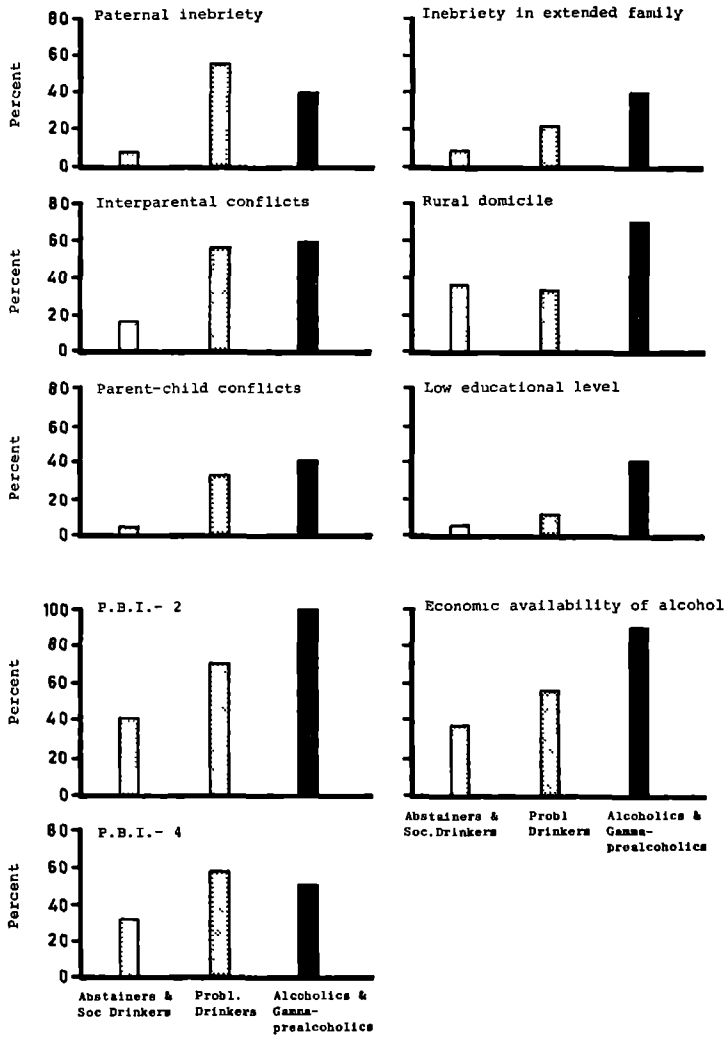


figure 5.4

Males older than 30 years predisposing factors

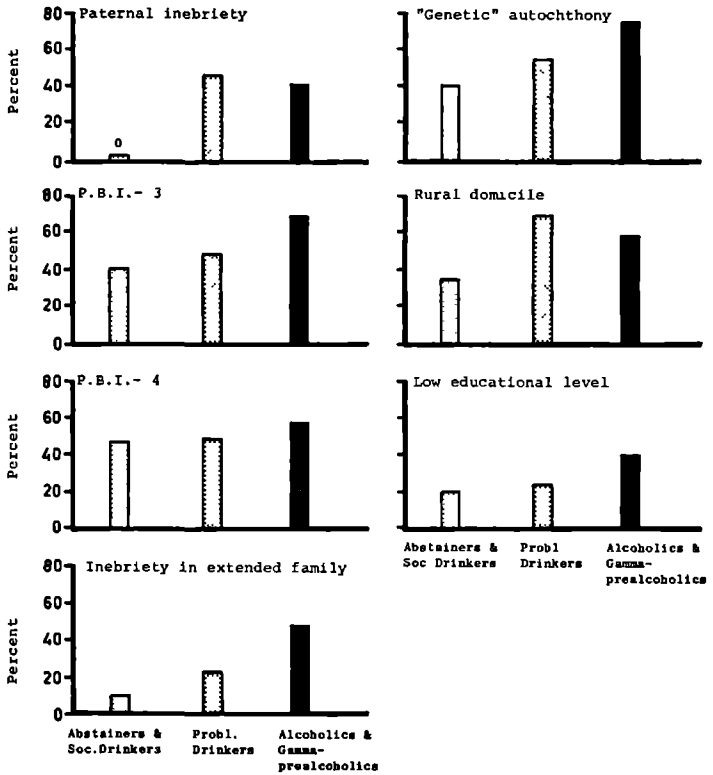


figure 55

All respondents predisposing factors.

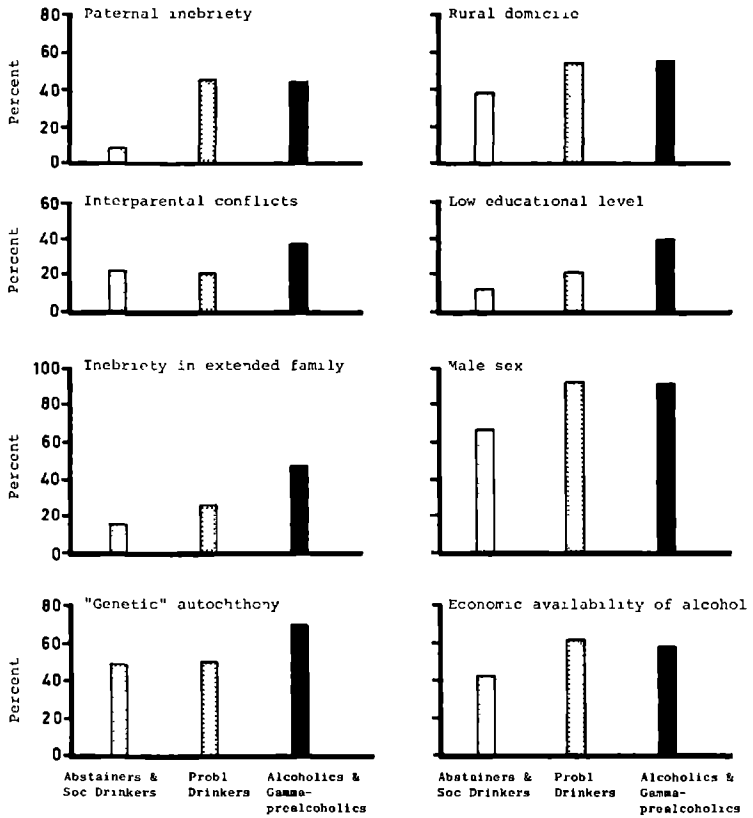


Table 5 10

Predisposing factors in percent per diagnostic category in survey A

Factor	Diagnostic category				γ^2	df	p
	Abstinence (N = 181)	Social Drinking (N = 371)	Problem Drinking (N = 83)	Prealcoholism & Gamma-prealcoholism & Alcoholism (N = 73)			
Familial inebriety	14	8	13	23	17 12	3	<0 005
Rural domicile	35	48	61	55	19 64	3	<0 005
Autochthony	80	75	89	85	9 92	3	<0 025
Male sex	25	49	69	92	107 89	3	<0 0001

5.3. RESULTS. PREVALENCES OF THE PERPETUATING FACTORS IN THE DIAGNOSTIC CATEGORIES

The perpetuating factors evaluated in this study can be categorized as shown in *table 5.11.*

Table 5 11

Perpetuating factors

I) *Actual factors in present home family, extended family and significant human relationships (microclimate)*

1 Marital conflicts
2 Conflicts with one's children
3 Low social status of respondent
4 Poor housing conditions
5 (Unmarried) civil state
6 Violation of one's confidence by significant relations
7 a Authority conflicts with superiors at work
7 b Authority conflicts with one's parents
7 c Authority conflicts with one's parents-in-law
8 Negative attitudes of relatives towards drinker's drinking

II) *Actual factors in macroclimate*

9 Social normativity
10 (Proneness to) social pressure to drink
11 Hospitality drinking
12 Stress at work
13 Sociocultural deprivation of autochthonous males
14 Sociocultural deprivation of heterochthonous females
15 (Low) religious involvement

III) *Actual endogenous or exogenous mental distress factors*

16 Anxiety
17. Suicidality
18. Boredom
19 Introversion
20 Dissatisfaction
21 Oral fixation or regression
22 Drugs

5.3.1. Actual factors in present family, parental family, extended family and significant human relations (microclimate)

Generally speaking, these factors can be considered as *stressful life events* (Bell et al., 1976). Little research has been performed that specifically evaluates the quantitative relationships among stressful life events, psychiatric disorders and alcoholism (Bell et al., 1976).

5.3.1.1. Marital conflicts

The prevalences of this factor are shown in *tables 5.16. & 5.17* and *figs. 5.6.-5.9.*

This factor can be interpreted as a *late sociocultural factor in the microclimate,* in the sense of "*escape drinking*" (Cahalan et al, 1969, Edwards, 1970, Gadourek, 1963, Jellinek, 1960, Jones, 1966, Kalant & Kalant, 1971) It implies a *circular causality,* since *marital conflicts* can be caused by alcoholism - through provocation of negative attitudes in the marital partner by the alcoholic's disruptive antisocial behavior - but reciprocally alcoholism can be perpetuated, provoked and stimulated by marital conflicts ("escape drinking")

The results are as expected, and in concordance with previous findings (Wever, 1971).

The findings reveal significant correlations in all subdivisions *except* "female respondents" and "males younger than 30 years". Still, even in this latter subdivision, this factor is considered an important perpetuating factor because of the strong correlations in the other subdivisions and the clear gradient (20% - 50% - 50%), cf. *table 5.17.* and *fig. 5.7.* Among female respondents χ^2 could not be applied because of the small N; still, because of the evident trend, this factor is considered an important one among females (*cf. table 5.16.*).

Appendix D shows the method used to evaluate this factor. This factor has been evaluated only among married respondents.

Marital conflicts occur frequently in marriages of alcoholic patients (Bailey, 1961; Bell et al, 1976, Jackson, 1962, Krimmel, 1973, Mac Donald, 1956; Paige et al, 1971; Whalen, 1953).

The *wife* of an alcoholic is not simply the object of mistreatment, but an active participant in the creation of marital conflicts (Krimmel, 1973, Whalen,

1953) The alcoholic's wife has been described as an insecure and dependent personality, who married in the expectation of meeting her dependency needs through a supposedly strong husband, when this husband also appears to be dependent, his wife's needs are increasingly frustrated with consequently increasing inadequate responses of the husband (Bailey, 1961, Krimmel, 1973) Some wives of alcoholics appear to hamper their alcoholic husbands' attempts to obtain help, seemingly needing to keep their husbands ineffectual, so that they keep feeling relatively stronger than their husbands, thus keeping their own inadequacies covered (Bailey, 1961, Paige et al, 1971) This latter statement is at variance with Jackson's finding, that only a minority of wives of alcoholics decompensated mentally when their husbands' alcoholism became inactive and that most of these wives were deeply involved in having their husbands achieve sobriety (Jackson, 1962) Disturbances in *all* family members may occur at the onset of sobriety, as a result of rigidity in family processes and interactions (Jackson, 1962) Mental breakdown has been observed in wives of alcoholics who attained sobriety (Mac Donald, 1956), as well as depression, phobia and psychosomatic illness such as gastrointestinal disorders and obesity, as well as alcoholism (Bailey, 1961) Sexual problems in wives of alcoholics have been observed, some as a reflection of disturbed relationships with their fathers, others as a reflection of the wives' frigidity, so that they would use their husbands' drinking as an excuse for avoiding intercourse (Bailey, 1961)

Whalen has described four *personality types among wives of alcoholics* (Whalen, 1953)

a) *masochism* the wife who needs to punish herself ("Suffering Susan"),
b) *control dominance* the wife who controls and dominates her husband ("Controlling Catherine"),
c) *vacillation* the wife who is characterized by wavering and self-doubt ("Wavering Winifred'),
d) *punitiveness* the wife who usually is a career woman, rivalrous and punishing towards men (Punitive Polly")

Husbands of female alcoholics are generally less patient and more likely to terminate the marriage than are the wives of male alcoholics This tendency is due to a) the greater tolerance of society towards masculine drinking, b) the wife's financial dependence on her husband's support, c) the wife's capacity to mother, once she senses her husband s illness (Bailey, 1961)

Bailey has described five *personality types among husbands of female alcoholics* (Bailey, 1961)

a) the long-suffering husband who spoils his child-wife,
b) the unforgiving self-righteous husband,
c) the husband who leaves furiously but comes running back,
d) the punitive, sadistic husband,
e) the dependent husband, who expected to find another mother in his wife, but who is hurt at finding that his wife has become as dependent as he, through her alcoholism

There have been, however, few studies on husbands of female alcoholics (Bailey, 1961)

Alcoholics and their partners tend to poison each other by the various toxic patterns of marital interaction (Paige et al, 1971) Hypothesis concerning marital conflicts in alcoholic marriages are many, but the most important factor is that a husband and wife in such a marriage have a *complementary pathology*, whether it was brought to the marriage or developed later (Krimmel, 1973) The spouse may derive considerable gratification from being the forgiver or the punisher The conscious wish generally is to help, yet these very behaviors contribute to the perpetuation of the problem, because the forgiving attitude has taught the alcoholic that forgiveness for intoxication can be obtained if he shows disease behavior or sufficient remorse, and the punishing attitude has relieved his guilt and shame towards excessive drinking (Krimmel, 1973)

The ways in which the male alcoholic tries to assert himself can result in *violence*, which may enable the alcoholic to feel that he is showing who is the man of the house (Krimmel, 1973) The alcoholic male may attempt to reestablish his place as the dominant male in the *sexual area* In this area the problems are compound since the alcoholic male may be impotent when drunk He may be potent when sober, but his wife, usually unable to forget, does not respond This is the point, where "*pathological marital jealousy*" develops (Krimmel, 1973) For the alcoholic female the sexual problem is different, since she can usually perform, and she may barter sex for alcohol (Krimmel, 1973)

Briefly, it can be stated, that marital conflicts in an alcoholic marriage are characterized by a relationship of complementary pathology, perpetuated by and perpetuating alcoholism, in such a situation maximal improvement can only be achieved by tackling both the alcoholism and the pathological complementary relationships between both partners and between *all* family members

5.3.1.2. Conflicts with one's children

The prevalences of this factor (variable no. 135) are shown in *tables 5.16 & 5 17.* Another item (variable no. 126) dealing with this factor, was omitted, because it measures this dimension analogically.

The results show no statistically significant correlations.

This factor can be interpreted as a *late sociocultural factor in the microclimate,* in the sense of "*escape drinking*" (Cahalan et al , 1969, Edwards, 1970, Gadourek, 1963, Jackson, 1962, Jellinek, 1960, Jones, 1966, Kalant & Kalant, 1971, Krimmel, 1973)

5.3.1.3. Low social status of respondent

The prevalences of this factor (variable no. L) are shown in *tables 5.16. & 5.17.* and *figs. 5.8.-5 9.*

Appendix D and *Appendix E* show the methods used to evaluate this factor.

The findings reveal significant correlations only among the subdivisions "all respondents" and "male respondents older than 30 years", and hence are in agreement with the observations of Cahalan et al., who found a correlation between *low* social status and heavy drinking (Albrecht, 1973; Cahalan, 1976; Cahalan & Cisin, 1968; Cahalan et al., 1969).

The findings are at variance with Knupfer's statement, that surveys of drinking patterns have consistently found increasing amounts of drinking with higher socio-economic status (Knupfer & Room, 1964), with statements concerning heavy drinking in both lower and upper social classes contrasting light drinking or abstinence in middle class (Lawrence & Maxwell, 1962), with the observation of low drinking prevalence and high abstinence prevalence among lowest social classes as compared to the remaining classes especially in wom-

en (Lawrence & Maxwell, 1962), and with the observation of high prevalence of heavy drinking and alcoholism among physicians - generally belonging to higher social classes - in Scotland (Murray, 1976).

This factor can be interpreted as a *late sociocultural factor in the microclimate*, in the sense of the "culture of poverty" as described in the section "rural domicile". It implies a circular causality, since low social status (high scores in *tables 5.16. and 5.17.*) can be caused by alcoholism (through impoverishment of the alcoholic), but reciprocally alcoholism can be perpetuated, provoked and stimulated by low social status in the sense of "*escape drinking*", (Bell et al. 1976; Cahalan et al., 1969; Edwards, 1970; Gadourek, 1963; Jellinek, 1960; Kalant & Kalant, 1971) to escape from "low social status" as a stressful life event.

5.3.1.4. Poor housing conditions

The prevalences of this factor (variable no. 8) are shown in *tables 5.16. & 5.17.* and *figs. 5.6. & 5.9.*

Appendix D shows the method used to evaluate this factor.

The findings only reveal significant correlations among the subdivision "all respondents". Among female respondents χ^2 could not be applied because of the small N; still, because of the evident trend (cf. *table 5.16.)*, this factor is considered an important one among females.

It can be argued theoretically, that alcoholism - through impoverishment of the alcoholic - can lead to poor housing conditions, but reciprocally can be provoked, stimulated and perpetuated by poor housing conditions once present (circular causality) in the sense of "escape drinking" to escape from those poor housing conditions as a "stressful life event" (Bell et al., 1976; Esser, 1965; Jellinek, 1960). Poor living and housing conditions of the manual worker, and absence of sports and recreation seemed to be at the root of excessive drinking in the Netherlands in the nineteenth century. Alcohol seemed to be the only means to combat the social strain to a certain extent (Esser, 1965).

5.3.1.5. (Unmarried) civil state

The prevalences of this factor (variable no. 6) are shown in *tables 5.16. & 5.17.*

Appendix D shows the method used to evaluate this factor.

The findings only reveal a significant correlation among the subdivision "all respondents". Therefore this factor is considered of *no* importance as a perpetuating factor. Moreover, the correlation among "all respondents" *opposes* the original expectation (R = —0.17546). This can be partially explained as follows:

a) Average age appears to *increase* from 27.4 among abstainers

through 32.9 in social drinkers and 37.5 in problem drinkers to 43.3 among alcoholics (and gamma-prealcoholics): it is evident that with increasing age there also occurs an increase in the prevalences of marriage and its counterparts i.e. common-law, divorce and widowed state;

b) Perhaps one must assume that married life *per se* may imply a stressful life event?

This factor can be interpreted as:

1) A *late sociocultural factor in the microclimate* in the sense of *"escape drinking"* (Cahalan et al., 1969; Criteria Committee, 1972; Jellinek, 1960). It implies, that married life would represent the least hazardous civil state as compared to common-law, divorced, widowed and single civil state.
2) A *psychological factor* in the sense of *introversion, solitariness,* and *low social participation* (Esser, 1967; Gadourek, 1963; Jellinek, 1960; Wexberg, 1951).

It implies a *circular causality*, since common-law, divorce and single civil state can be caused by alcoholism (through the alcoholic's disruptive antisocial behavior impeding his ever marrying or destroying his actual marriage and possibly leading him into common-law unions), while reciprocally alcoholism can be perpetuated, provoked and stimulated by unmarried civil state.

5.3.1.6. Violation of confidence by relatives & significant relations

The prevalences of this factor are shown in *tables 5.16. & 5.17.* and *figs. 5.6.-5.7.*

Appendix D shows the method used to evaluate this factor.

This factor can be interpreted as a *late sociocultural factor in the microclimate,* in the sense of "escape drinking", to escape from violation of one's confidence as a stressful life event (Bell et al., 1976; Cahalan et al., 1969; Edwards, 1970; Gadourek, 1963; Jellinek, 1960; Kalant & Kalant, 1971; Milt, 1969).

The stressful life events included in this factor are highly interpersonal or social, and involve maladaptive interpersonal or social behaviors.

This factor implies a *circular causality*, since violation of one's confidence can be caused by one's alcoholism, while at the same time alcoholism can be perpetuated, provoked and stimulated by the violation of one's confidence by significant relations.

The results are as anticipated, and reveal significant correlations among all subdivisions studied.

However, because among "male respondents older than 30 years" and among "all female and male respondents" problem drinkers have a lower prevalence of this factor as compared to abstainers & social drinkers (and to alcoholics & gamma-prealcoholics), this factor will be omitted from the conjoint evaluation of all factors for both these subdivisions. Reason: perhaps this factor stimulates both alcoholism and abstinence among the subdivisions mentioned.

5.3.1.7.1. Authority conflicts with superiors at work

5.3.1.7.2. Authority conflicts with one's parents

5.3.1.7.3. Authority conflicts with one's parents-in-law

The prevalences of these factors (variables no. 128, 129, 130) are shown in *tables 5.16. & 5.17.*

Appendix D shows the methods used to evaluate these factors.

The findings reveal significant correlations for all subdivisions except female respondents. These correlations are debilitated, however, by:

a) The fact, that variable no. 128 was only applicable to respondents having employment;
b) The fact, that variable no. 130 was only applicable to married respondents;
c) The fact, that variables no. 128, 129 and 130 had "not applicable" responses exceeding 5%.

These factors can be interpreted as *late sociocultural factors in the microclimate and macroclimate* in the sense of *'escape drinking"*, to escape from authority conflicts (with parents, parents-in-law, supervisors at work) as stressful life events (Bell et al, 1976, Cahalan et al, 1969, Jellinek, 1960, Milt, 1969, Paige et al, 1971)

A circular causality is implied, since authority conflics can be caused by a person's alcoholism, while reciprocally alcoholism can be perpetuated, provoked and stimulated by authority conflicts

5.3.1.8. Negative attitudes of relatives towards drinker's drinking habits

The prevalences of this factor are shown in *tables 5.16 & 5.17.*

Appendix D shows the method used to evaluate this factor.

This factor can be interpreted as a *late sociocultural factor in the microclimate* in the sense of *"escape drinking"*, to escape from these negative relatives' attitudes implying a stressful life event (Bell et al, 1976, Cahalan et al, 1969, Edwards, 1970, Jellinek, 1960, Kalant & Kalant, 1971, Milt, 1969)

It implies a *circular causality*, since negative relatives' attitudes evidently can be caused by a person's alcoholism, reciprocally alcoholism can be perpetuated, provoked and stimulated by negative attitudes of relatives towards the drinker's drinking habits

However, because in this circular causality the shift "alcoholism (or its precursor stages) → negative relatives' attitudes towards drinker's drinking habits" is considered more important than the shift in the opposite direction, this factor is *excluded* from the conjoint evaluation of all factors in this study to be described below.

The findings reveal significant correlations among all subdivisions evaluated, except "female respondents" (χ^2 not applicable because of

the small N). However, even among females the trend is clear and constistent with the alcoholismic gradient.

5.3.2. Actual factors in macroclimate

Generally speaking, these factors can be considered as either *factors which positively facilitate the development of excessive drinking* (social normativity; proneness to social pressure to drink; hospitality drinking) or as *stressful life events* (stress in the working situation; sociocultural deprivation of autochthonous males; sociocultural deprivation of heterochthonous females) (Bell et al., 1976).

5.3.2.1. Social normativity

The prevalences of this factor in *survey B & C* are shown in *tables 5.16. & 5.17.*

Mean factor scores of this factor in *survey A* are shown in *table 5.19.* Factor analysis of this factor is shown in *table 5.12.*

Table 5.12

Factor analysis of the factor "social normativity"

Variable No.	Content of variable	Factor loading (survey B & C)	Factor loading (survey A)
46	What would you think if someone of your family would be drunk occasionally?	0.7756	0.7272
47	What would you think if someone of your family would be „tipsy" occasionally?	0.7090	0.6730
48	What would you think if someone of your family would be drunk every week?	0.6983	0.6225
49	What would you think if someone of your family would have a drink with the daily meals?	0.4555	0.4674
50	What would you think if someone of your family would be drinking regularly without ever getting really drunk?	0.4300	0.5664
51	What would you think if someone of your family would be getting drunk every night?	0.5963	0.6023
52	What would you think if someone of your family on principle never would drink?	0.4389	0.0709

Higher scores on this factor express *rejecting intolerant attitudes towards (excessive) alcohol consumption and intoxication* by one's relatives, while lower scores express the opposite attitudes.

Appendix D shows the method used to evaluate this factor in survey B & C; below median scores are considered to characterize this factor, whereas median factor score is 500.

In *survey A* the results are as anticipated: *lower* factor scores among alcoholics & (gamma-)prealcoholics ($R = -0.1208$, $p = 0.001$).

In *survey B & C*, however, the findings reveal *no* significant correlations among the subdivisions studied.

This factor can be interpreted as a *sociocultural factor in the macroclimate* (Beaubrun, 1971, Gadourek, 1963, Jansen, 1969, Plaut, 1967): tolerant attitudes towards (excessive) drinking by significant others is considered conducive to tolerant attitudes to one's own drinking excesses and hence to one's progression on the alcoholismic gradient

It implies a *circular causality*, since a person's *tolerant attitudes* towards (excessive) drinking by significant others can be caused by the person s alcoholism (through the alcoholic s approval of his own excessive drinking pattern when he is confronted with this pattern in relatives), while reciprocally a person s alcoholism can be perpetuated, provoked and stimulated by his/her tolerant attitudes towards excessive drinking

The results in this study are inconsistent: in *survey A* there is a clear correlation between the alcoholismic gradient and tolerant attitudes towards drinking excesses, while in *survey B & C* there is no correlation. The reason for this discrepancy is as yet not clear.

5.3.2.2. (Proneness to) social pressure to drink/social dependence

The prevalences of this factor in *survey B & C* are shown in *tables 5.16. & 5.17.* and *figs 5.6.-5.9.*

The prevalences of this factor in *survey A* are shown in *table 5.20.*

Appendix D shows the method used to evaluate this factor.

The findings reveal significant correlations among *all* subdivisions evaluated.

This factor can be interpreted as

1) a *sociocultural factor in the macroclimate*, reflecting peer group pressure exerted by friends on an individual to drink (excessively) whenever this is the peer group habit,
2) a *psychological factor*, reflecting the individual's over-readiness to give in to this peer group pressure (social dependence)

Like the preceding factor (social normativity), this one is a factor which positively facilitates progression on the alcoholismic gradient Though "(proneness to) social pressure to drink" may diminish mutual reserve and stimulate social contact (Bales, 1962, Heath, 1962, Horwitz et al , 1967) - a positive characteristic of this factor - its ultimate consequence is definitively a negative one, that is alcoholism

5.3.2.3. Hospitality drinking

The prevalences of this factor (variable no. 136) in *survey B & C* are shown in *tables 5.16. & 5.17.*

The prevalences of this factor in *survey A* are shown in *table 5 20*

The findings reveal *no* statistically significant correlations except in the subdivision "male respondents older than 30 years". However, even in this subdivision, the prevalence of the factor "hospitality drinking" among problem drinkers is lower than among the combined group of abstainers and social drinkers though also lower than among alcoholics. Moreover, as may be seen in *Appendix A*, this factor was not adequately operatized, since it constitutes one out of seven possible answers for a single question. Therefore this factor - though separately it may play a significant role - is *not* considered as an important perpetuating factor in the conjoint evaluation of all factors in this study.

The factor can be interpreted as a *sociocultural factor in the macroclimate* the learning of alcohol consumption in certain social settings facilitating an individual's own (excessive) drinking, such as (excessive) drinking in the host-guest relationship in order to diminish mutual reserve and to achieve mutual contact (Bales, 1962, Heath, 1962, Horwitz et al , 1967)

In this view, alcoholism and its precursor stages imply an *exaggeration* of prevailing drinking patterns This implies a *circular causality*, since "hospitality drinking" can be caused by a person's alcoholism, while reciprocally alcoholism can be perpetuated, provoked and stimulated by prevailing 'hospitality drinking patterns" in a community

Heath's description of drinking patterns (hospitality drinking, drinking for the purpose of social cohesion) among the Bolivian Camba, a mixed Indian-White population, is in marked agreement with drinking customs among Arubans, also a mestizo population (Heath, 1962) The inclination to drinking for purely utilitarian or convivial purposes (for social cohesion or hospitality) has been associated with problem drinking e g in the Irish, Irish-Americans, French and Anglosaxon Protestants in the U S A (Bacon, 1973, Bales, 1962, Beaubrun, 1971)

On the other hand "integrated drinking" (i e drinking associated with religious or ritual ceremonies) might be a factor in diminishing problem drinking (Bacon, 1973)

5.3.2.4. Stress at work

The prevalences of this factor are shown in *tables 5.16 & 5.17.*

Appendix D shows the method used to evaluate this factor.

The findings for *female respondents* were not suitable for statistical analysis because of the very low figures per diagnostic category (cf. *table 5.16.).*

The findings among the remaining subdivisions (cf. *table 5.17.*) reveal *no* significant correlations.

Therefore this factor *cannot* be considered as an important perpetuating factor in this study.

The factor can be interpreted as a *sociocultural factor in the macroclimate.*

5.3.2.5. Sociocultural deprivation of autochthonous males

The prevalences of this factor (variable no. 142) in *survey B & C* are shown in *table 5.17.* and *figs. 5.7. & 5.9.*

The findings in *survey B & C* reveal significant correlations for the subdivisions "male respondents younger than 30 years", "male respondents older than 30 years", and "all male respondents". However, this factor was only tested among *autochthonous* (Aruban) males, not among females or heterochthonous males, in survey B & C.

Because among "male respondents older than 30 years" the prevalence of this factor in problem drinkers is *lower* than among the combined group of abstainers and social drinkers, this factor is only considered an important perpetuating factor among "males younger than 30 years" and "all male respondents".

This factor can be interpreted as a *sociocultural factor in the macroclimate.* It implies a *circular causality,* since (perception of) „deprivation of autochthonous males" can be caused by an autochthonous male's alcoholism through the induction of disapproval in autochthonous females of the autochthonous male's role, reciprocally an autochthonous male's alcoholism can be perpetuated, provoked and stimulated by (perception of) "deprivation of autochthonous males by autochthonous females" in the sense of "*escape drinking*" to escape from this deprivation perceived as a stressful life event (Bell et al, 1976, Cahalan et al, 1969, Jellinek, 1960).

5.3.2.6. Sociocultural deprivation of heterochthonous females

The prevalences of this factor (variable no. 143) in *survey B & C* are shown in *table 5.16.*

The findings in *table 5.16.* are not suitable for statistical analysis because of the very low figures per diagnostic category, though the *trend* is *consistent* with the alcoholismic gradient. Moreover, this factor was only evaluated among *heterochthonous females.* Therefore, though this factor may separately play a significant role, it is *not* considered an important perpetuating factor in the conjoint evaluation of all factors in this study.

The factor can be interpreted in analogous terms to the preceding one.

5.3.2.7. (Low) religious involvement

The prevalences of this factor are shown in *tables 5.16. & 5.17.*

Appendix D shows the method used to evaluate this factor.

The findings reveal *no* significant correlations among any of the subdivisions evaluated.

"*Low* religious involvement" was found to correlate with the alcoholismic gradient in other studies, especially when it reflects "low religious involvement" in connection with *Protestantism* (Calahan et al, 1969, Gadourek, 1963, Wallace, 1972)

"*High* religious involvement" connected with *Roman Catholicism*, however, was found to correlate with the alcoholismic gradient (Calahan et al, 1969, Gadourek, 1963, Knupfer & Room, 1964), whereas "*high* religious involvement"*per se* may be found correlated with the alcoholismic gradient as a result of the religious indoctrination by Alcoholics Anonymous (Wahl, 1956) or as a reflection of the *symptom* "vague religious desires" in chronic alcoholism (Jellinek, 1962)

It is evident, from the results in *tables 5.16. & 5.17.*, that this factor cannot be considered as an important perpetuating factor in this study.

5.3.3. Actual endogenous or exogenous mental distress factors

Generally speaking, these factors can be considered as either *(congenital or acquired) psychological factors* or as *late sociocultural factors in the macroclimate;* in both instances they imply either *internal stressful life events* from which pathological drinkers try to escape, or *external stressful life events,* or *mental distress factors caused by pathological drinking.*

5.3.3.1. Anxiety

The prevalences of this factor (variable no. 77) are shown in *tables 5.16 & 5.17* and *figs. 5.7-5.9.*

The findings reveal significant correlations among *all* subdivisions evaluated, except "female respondents", though even there the *trend* is consistent with the alcoholismic gradient. However, for this reason this factor *cannot* be considered as an important perpetuating factor in the conjoint evaluation of all factors among females in this study.

The factor can be interpreted as a (congenital or acquired) *psychological factor* (Criteria Committee, 1972, Gadourek, 1963, Horton, 1943, Jellinek, 1960, Milt, 1969, Sayres, 1956, Wahl, 1956)

Moreover, continuously increasing doses of ethanol may *produce* anxiety, (Criteria Committee, 1972), and anxiety is one of the symptoms in alcohol withdrawal syndromes (Cf Chapter 1) It implies a *circular causality,* since anxiety can be caused by alcoholism (disruptive anti-social behavior in the intoxicated state → confrontation with the consequences of this behavior in the sober state → reintroduction of anxiety concerning these consequences), while

reciprocally alcoholism can be perpetuated, provoked and stimulated by anxiety as an internal stressful life event from which the drinker tries to escape (Bacon, 1973, Bell et al, 1976, Horton, 1943, Jellinek, 1960, Milt, 1969, Sayres, 1956, Wahl, 1956) Clinical experience has suggested, that alcohol is an effective external reducer of anxiety loneliness, guilt, sexual inhibition, social inhibition, impaired self-esteem, and other unpleasant emotional states And in animal studies it was also shown to be an effective fear reducer (Wahl, 1956)

5.3.3.2. Suicidality

The prevalences of this factor are shown in *tables 5.16. & 5.17.* and *figs. 5.6 -5 9.*

Appendix D shows the method used to evaluate this factor.

The factor can be interpreted as a (congenital or acquired) *psychological factor* (Cahalan et al, 1969, Gadourek, 1963, Jellinek, 1960, Palola et al, 1962, Tinklenberg, 1973)

Moreover, alcohol as a CNS-depressant may generate depression and hence suicidality (Jones et al, 1970) Depression (and hence suicidality), is one of the symptoms in alcohol withdrawal syndromes though less frequent than anxiety or hyper-excitability (the opposite of depression) (cf Chapter 1)

This factor implies a *circular causality,* since depression and suicidality can be caused by alcoholism (Jones et al, 1970, Palola et al, 1962), while reciprocally alcoholism can be perpetuated, provoked and stimulated by (depression and) suicidality as an internal stressful life event from which the drinker tries to escape (Cahalan et al, 1969, Jellinek, 1960, Milt, 1969, Palola et al, 1962, Tinklenberg, 1973)

Alcoholism was considered as the *cause* in 23 0% of attempted suicides and 31 4% of completed suicides (Palola et al, 1962) Suicidal behavior (both thoughts and attempts) was considered to have appeared *before* the onset of alcoholism (using the inception of loss-of-control as "pathognomonic sign" for alcoholism) in a majority of interviewed alcoholics, and hence it was concluded that alcoholism can be viewed as a *substitute* for, i e the *consequence* of suicidality It was hypothesized that excessive drinking would give an escape from the individual s tendency to definite self-destruction through the oblivion and relative pleasure of drinking (Palola et al, 1962) At some later point, after the onset of addiction, the alcoholic would have the experience of „hitting the bottom" the alcoholic then realises that everything is hopelessly lost and that even alcohol is not able to give adequate relief from distress any longer At that point, the alcoholic may seek some definite solution either external help (e g from the medical profession or from AA), or suicide Among alcoholics most completed suicides were found *after* the onset of alcohol addiction, while attempted suicides were more prevalent in prealcoholics, i e *before* the onset of addiction (Palola et al, 1962) Depression was invariably present in alcoholics who attempted or committed suicide (Palola et al, 1962) Precipitating events to attempted or committed suicide among alcoholics, were disrupted interpersonal relationships such as divorce, separation or death of a loved one, serious physical illness such as liver cirrhosis, discharge from jobs, and low levels of social integration in the sense of anomia (Palola et al, 1962)

The findings reveal significant correlations among *all* subdivisions evaluated, except "female respondents" and "males younger than 30 years". This may imply that this factor is only significant after the age of 30 among males. Among female respondents χ^2 could not

be applied because of the small N; still, because of the evident trend, this factor is considered an important one among females (cf. *table 5.16.*).

5.3.3.3. Boredom

The prevalences of this factor (variable no. 101) are shown in *tables 5.16. & 5.17.* and *figs. 5.7.-5.9.*

This factor can be interpreted as.

a) A (congenital or acquired) *psychological factor* reflecting a passive form of self-alienation (Van der Does de Willebois, 1965)
b) A *sociocultural factor in the macroclimate*, reflecting lack of recreational possibilities in a community
c) A *sociocultural factor in the macroclimate*, reflecting lack of social integration (anomia)

This factor implies a *circular causality*, since boredom can be caused by alcoholism (through the alcoholic's loss of interest in social activities except alcohol-related activities), while reciprocally alcoholism can be perpetuated, provoked and stimulated by boredom as an internal or external stressful life event from which the drinker tries to escape (Cahalan et al, 1969, Gadourek, 1963, Jellinek, 1960, Milt, 1969, Van der Does de Willebois, 1965).

The findings reveal significant correlations among *all* subdivisions evaluated, *except* female respondents. Therefore this factor *cannot* be considered as an important perpetuating factor in the conjoint evaluation of all factors among female respondents in this study.

5.3.3.4. Introversion

The prevalences of this factor (variable no. 106) are shown in *tables 5.16. & 5.17.* and *figs. 5.7.-5.9.*

This factor can be interpreted as

a) A (congenital or acquired) *psychological factor* (Esser, 1967; Gadourek, 1963; Jellinek, 1960, Wexberg, 1951) in the sense of schizoid tendencies
b) A *sociocultural factor* in the macroclimate, reflecting lack of social integration (anomia)

It implies a *circular causality*, since introversion can be caused by alcoholism (through the alcoholic's disruptive antisocial behavior thus impeding the development of socialization skills), while reciprocally alcoholism can be perpetuated, provoked and stimulated by introversion as an internal stressful life event from which the drinker tries to escape (Jellinek, 1960, Milt, 1969, Wexberg, 1951)

The findings reveal significant correlations among *all* subdivisions evaluated, *except* female respondents, though even there the *trend* is *consistent* with the alcoholismic gradient. Therefore this factor *cannot* be considered as an important perpetuating factor in the conjoint evaluation of all factors among female respondents in this study. The results are in agreement with previous reports concerning schizoid traits among Aruban alcoholics (Turfboer, 1957; Wever, 1971).

5.3.3.5. Dissatisfaction

The prevalences of this factor are shown in *tables 5.16. & 5.17.* and *figs 5 7.-5 9.*

Appendix D shows the method used to evaluate this factor.

This factor can be interpreted as

a) A (congenital or acquired) *psychological factor*

b) A *late sociocultural factor in the microclimate,* in the sense of *"escape drinking"* from an internal and external stressful event from which the drinker tries to escape (Cahalan et al, 1969, Edwards, 1970, Gadourek, 1963, Jellinek, 1960, Kalant & Kalant, 1971, Milt, 1969)

It implies a *circular causality,* since dissatisfaction can be caused by alcoholism (through impairment of health, reduction of economic potential and impoverishment of the alcoholic), while reciprocally alcoholism can be perpetuated, provoked and stimulated by dissatisfaction ("escape drinking")

The findings reveal significant correlations among *all* subdivisions evaluated, *except* female respondents, though even there the *trend* is *consistent* with the alcoholismic gradient. Therefore this factor *cannot* be considered, as an important perpetuating factor in the conjoint evaluation of all factors among female respondents in this study.

5.3.3.6. Oral fixation or regression

The prevalences of this factor are shown in *tables 5.16. & 5 17.* and *figs 5.7.-5 9*

Factor analysis of this factor is shown in *table 5.13.*

Table 5 13

Factor analysis of the factor „oral fixation or regression"

Variable No	Content of variable	Factor loading
21	How many cups of coffee or tea do you drink daily?	0 4667
22	How often do you eat biscuits, pies or sweets?	0 5591
23	Are there certain times at which you tend to eat (more) sweets?	0 4720
24	What do you usually smoke?	0 7730
25	How much do you usually smoke a day?	0 7956

This factor can be interpreted as follows

a) As presumably covering the Freudian theory, i e *fixation* in the oral phase of the development of libido, representing *underdevelopment* of libido, or

regression to less mature stages of libido, caused by emotional deprivation in childhood or later in life, both fixation and regression would lead to a proliferation of oral activities aimed at satiation of libido including excessive eating, drinking and smoking This represents a (congenital or acquired) *psychological factor* (Edwards, 1970 Gadourek, 1963, Milt, 1969)

b) As presumably reflecting a *social habit formation* i e excessive use of coffee, tea and sweets as well as excessive smoking - oral activities which may be *consequences* of excessive drinking, because it is a popular belief, that coffee and smoking can combat alcoholic intoxication and hangover (Gadourek, 1963) This represents a *sociocultural factor in the macroclimate*

c) As presumably reflecting the *virility complex*, i e smoking and drinking to symbolize *manhood* and *adult life* (Gadourek, 1963, Hoetink, 1957, Mc Cord & Mc Cord, 1962)

d) As presumably reflecting an *acquired need of stimulant drugs* (e g caffeine, nicotine) to combat the sedative effects of alcohol

Hence it is evident, that this factor truly implies a *perpetuating factor* (and not a solely predisposing factor) it may be provoked by alcoholism (cf sub b) and d)), and once present it may perpetuate alcoholism (cf sub a) and c))

The findings reveal significant correlations among *all* subdivisions evaluated, *except* female respondents. Therefore this factor *cannot* be considered as an important perpetuating factor in the conjoint evaluation of all factors among female respondents in this study.

The main sources of this factor ("oral fixation or regression") are the variables concerning *coffee/tea consumption* and *smoking* as shown in *table 5 14*

Table 5 14

*Distribution (in percent) of above median * score for daily consumption of coffee or tea (variable no 21), frequency of consumption of sweets (variable no 22), frequency of proneness to consumption of sweets (variable no 23), and quantity of daily smoking (variable no 25) in survey B & C*

Diagnostic category	N	Variable No 21	22	23	25
Abstinence	28	14	43	39	7
Social Drinking	41	32	24	24	24
Problem Drinking	24	42	29	25	42
Alcoholism (& Gamma)	131	60	37	19	66
		$\chi^2 = 25\ 57$ df = 3 p <0 005	$\chi^2 = 6\ 15$ df = 3 n s	$\chi^2 = 5\ 38$ df = 3 n s	$\chi^2 = 44\ 76$ df = 3 p <0 005

* In all 4 variables median score is 1

Though in survey B & C "oral fixation or regression" appeared as *one* factor in factor analysis, in survey A *two* factors were found

and confirmed; both these factors will be described in the next section.

5.3.3.6.1. Oral fixation or regression I ("excessive smoking")

5.3.3.6.2. Oral fixation or regression II ("excessive consumption of sweets")

Mean factor scores for both these factors, evaluated in survey A, are shown in *table 5.19.*

Factor analysis of these factors is shown in *table 5.15.*

Table 5.15

Factor analysis of the factors "oral fixation or regression I" and "oral fixation or regression II" in survey A

Variable No	Content of variable	Factor loading
	Oral fixation or regression I	
24	What do you usually smoke?	0 9270
25	How much do you usually smoke a day?	0 9186
	Oral fixation or regression II	
22	How often do you eat biscuits, pies or sweets?	0 7975
23	Are there certain times at which you tend to eat (more) sweets?	0 7787

The results indicate, that "*excessive smoking*" is *positively* correlated with the alcoholismic gradient, whereas "*excessive consumption of sweets*" is *negatively* correlated; the latter finding is at variance with the original expectation.

5.3.3.7. Drugs

The prevalences of this factor in survey B & C are shown in *tables 5.16. & 5.17.* and *fig. 5.7.*

Appendix D shows the method used to evaluate this factor.

The prevalences of this factor in survey A are shown in *table 5.20.*

The findings reveal *no* significant correlations, *except* in the subdivision "male respondents younger than 30 years". Though not statistically significant, among "female respondents" the *trend* is *consistent* with the alcoholismic gradient.

The results reflect the fact, that use of psychoactive drugs is a relatively recent phenomenon in Aruba: from Police reports it can be deduced, that drugs are playing a role of some significance only after 1960. Hence it is understandable, why older alcoholics and problem drinkers apparently are not affected in contrast to those younger than 30 years.

According to Isbell, drug dependence is frequently mixed, and (excessive) use of one drug tends to lead to (excessive) use of another (Isbell, 1970).

Table 5 16

Perpetuating factors in percent per diagnostic category for female respondents.

Factor	Diagnostic category		χ^2	df	p
	Abstinence & Social Drinking (N = 24)	Problem Drinking & Alcoholism & Gamma-prealcoholism (N = 14)			
Marital conflicts[1]	0	39	—	—	—
Conflicts with one's children[2]	13	11	—	—	—
Low social status of respondent	54	57	0 00	1	n s
Poor housing conditions	8	50	—	—	—
(Unmarried) civil state	67	71	0 00	1	n s
Violation of one's confidence	46	79	4 17	1	<0 05
Authority conflicts with superiors at work[3]	0	0	—	—	—
Authority conflicts with parents	4	21	—	—	—
Authority conflicts with parents-in-law[4]	0	0	—	—	—
Relatives' negative attitudes to one's drinking	0	64	—	—	—
Social normativity	33	57	1 86	1	n s.
(Proneness to) social pressure to drink	21	71	11 46	1	<0 005
Hospitality drinking	42	36	0 30	1	n s.
Stress at work[5]	25	0	—	—	—
Sociocultural deprivation foreign females[6]	0	40	—	—	—
(Low) religious involvement	100	100	0 00	1	n s.
Anxiety	50	64	0 47	1	n s.
Suicidality	0	29	—	—	—
Boredom	50	64	0 47	1	n.s.
Introversion	21	36	—	—	—
Dissatisfaction	13	36	—	—	—
Oral fixation or regression	58	57	0 00	1	n s.
Drugs	0	14	—	—	—

χ^2 was not applied for those factors where the figures were too low for statistical analysis.

[1] Eleven abstainers & social drinkers; thirteen others.
[2] Eight abstainers & social drinkers; nine others.
[3] No respondents.
[4] Ten abstainers & social drinkers; eleven others.
[5] Eight abstainers & social drinkers; three others.
[6] Two abstainers & social drinkers; five others.

figure 5.6.
Females perpetuating factors.

Table 5 17

Perpetuating factors in percent per diagnostic category
a) male respondents younger than 30 years (= M <30)
b) male respondents older than 30 years (= M >30)
c) all female and male respondents (= F + M)

Factor	Diagnostic category			R	p
	Abstinence & Social Drinking	Problem Drinking	Alcoholism & Gamma prealcoholism		
Marital conflicts[1]					
M <30	20	50	50	0 29752	n s
M >30	12	15	58	0 34420	0 0000
F + M	9	19	55	0 38916	0 0000
Conflicts with one's children[2]					
M <30	0	0	0	—	—
M >30	15	10	17	0 02930	n s
F + M	13	9	17	0 04001	n s
Low social status of respondent					
M <30	40	67	50	0 14463	n s
M >30	35	46	68	0 20909	0 0014
F + M	44	50	66	0 21500	0 0007
Poor housing conditions					
M <30	4	11	10	0 06198	n s
M >30	10	0	17	0 07598*	0 0679
F + M	7	8	20	0 12109	0 0059
(Unmarried) civil state					
M <30	80	89	70	—0 04545	n s
M >30	35	15	38	0 06368	n s
F + M	62	50	43	—0 17546	0 0048
Violation of one's confidence					
M <30	24	44	70	0 38017	0 0065
M >30	45	15	52	0 12002	0 0470
F + M	38	25	57	0 20512	0 0012
Authority conflicts with superiors at work[3]					
M <30	0	0	20	0 18656	0 0459
M >30	5	0	19	0 11130	0 0281
F + M	3	0	18	0 14952	0 0022
Authority conflicts with parents					
M <30	4	11	40	0 27273	0 0057
M >30	0	0	10	0 07201*	0 0302
F + M	3	8	13	0 09144*	0 0104
Authority conflicts with parents-in-law[4]					
M <30	0	0	33	0 29630	0 0000
M >30	0	9	14	0 08377*	0 0744
F + M	0	8	13	0 10254	0 0209
Relatives' negative attitudes to one's drinking					
M <30	20	67	80	0 55165	0 0002
M >30	25	92	86	0 28248	0 0000
F + M	15	75	85	0 61041	0 0000

Social normativity					
M <30	60	56	60	—0 01240	n s
M >30	35	62	50	0 04285	n s
F + M	44	63	50	0 04448	n s
(Proneness to) social pressure to drink					
M <30	32	89	90	0 56612	0 0001
M >30	35	54	80	0 27653	0 0000
F + M	29	67	80	0 45225	0 0000
Hospitality drinking					
M <30	40	44	60	0 15289	n s.
M >30	30	23	44	0 11605	0 0495
F + M	38	33	44	0 06920	n s
Stress at work					
M <30	0	17	20	—	—
M >30	26	10	19	—	—
F + M	18	12	18	0 01111	n s.
Sociocultural deprivation Aruban males[c]					
M <30	33	44	80	0 37750	0 0123
M >30	25	18	43	0 12824	0 0359
M (= Aruban males)	30	30	46	0 14224	0 0292
(Low) religious involvement					
M <30	80	89	100	0 16529	0 0627
M >30	90	92	95	0 03333	n s.
F + M	90	92	96	0 05931*	0 0354
Anxiety					
M <30	16	56	40	0 27893	0 0259
M >30	20	23	63	0 30192	0 0000
F + M	29	38	62	0 30891	0 0000
Suicidality					
M <30	4	11	10	0 06198	n s
M >30	0	0	14	0 09819*	0 0130
F + M	1	4	15	0 13098	0 0006
Boredom					
M <30	16	22	80	0 46694	0 0007
M >30	20	23	47	0 18409	0 0045
F + M	29	25	51	0 21995	0 0005
Introversion					
M <30	12	22	40	0 22521	0 0378
M >30	5	15	37	0 20234	0 0011
F + M	13	17	37	0 23230	0 0001
Dissatisfaction					
M <30	4	44	40	0 36570	0 0020
M >30	10	15	34	0 15850	0 0074
F + M	9	25	35	0 23724	0 0000
Oral fixation or regression					
M <30	24	89	60	0 43388	0 0028
M >30	55	62	90	0 23408	0 0000
F + M	45	71	85	0 35587	0 0000
Drugs					
M <30	4	22	40	0 30372	0 0041
F + M	5	8	5	—0 00913	n s
M >30	3	17	8	0 02966	n s

R = Kendall's Tau C [c] R not significant if —0 10 <R<+0 10

M <30 *25* abstainers & social drinkers, *9* problem drinkers, *10* alcoholics & gamma-prealcoholics

M >30 : *20* abstainers & social drinkers, *13* problem drinkers, *109* alcoholics & gamma-prealcoholics.
F + M : *69* abstainers & social drinkers, *24* problem drinkers, *131* alcoholics & gamma-prealcoholics.

[1], [2], [3], [4], [5], [6]. The numbers (N) of respondents for these factors are shown in *table 5.18.*

Table 5.18.

Numbers (N) of respondents per diagnostic category for six perpetuating factors.

Factor	Diagnostic category		
	Abstinence & Social Drinking	Problem Drinking	Alcoholism & Gamma-prealcoholism
Marital conflicts			
M <30	5	2	4
M >30	17	13	85
F + M	33	16	101
Conflicts with one's children			
M <30	3	0	2
M >30	13	10	69
F + M	24	11	79
Authority conflicts with superiors at work			
M <30	11	6	10
M >30	19	10	86
F + M	38	17	98
Authority conflicts with parents-in-law			
M <30	5	1	3
M >30	14	11	71
F + M	29	13	84
Stress at work			
M <30	11	6	10
M >30	19	10	86
F + M	38	17	98
Sociocultural deprivation Aruban males			
M <30	21	9	10
M >30	12	11	96
M (= Aruban males)	33	20	106

figure 5.7.

Males younger than 30 years perpetuating factors.

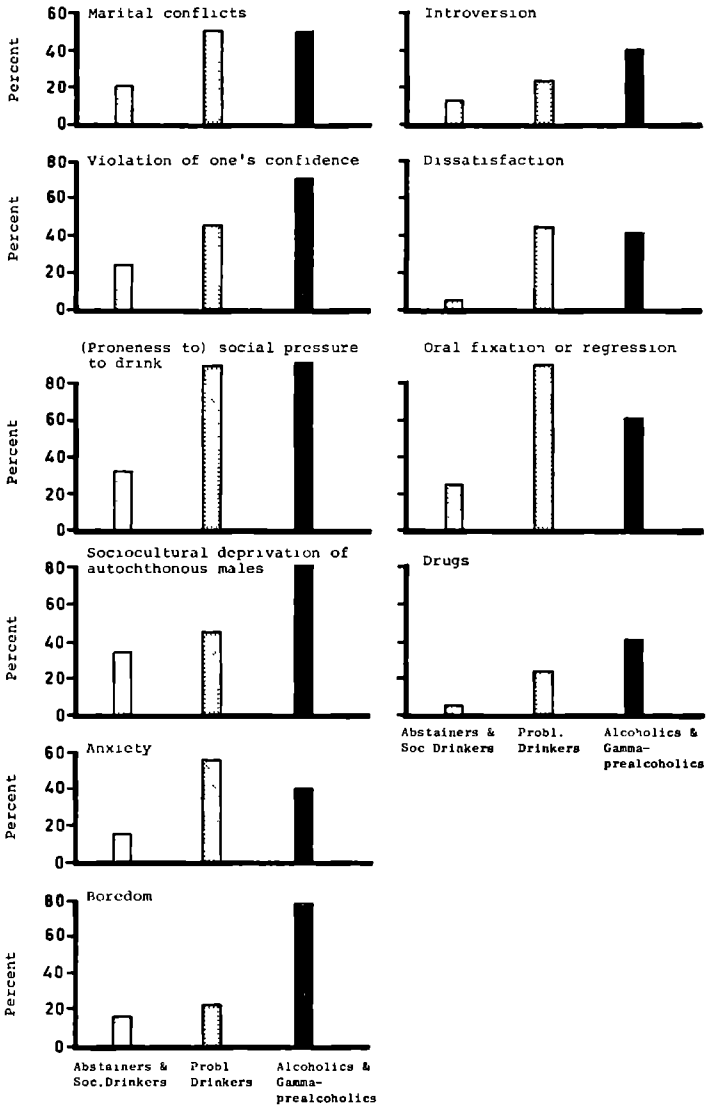


figure 5 8

Males older than 30 years perpetuating factors.

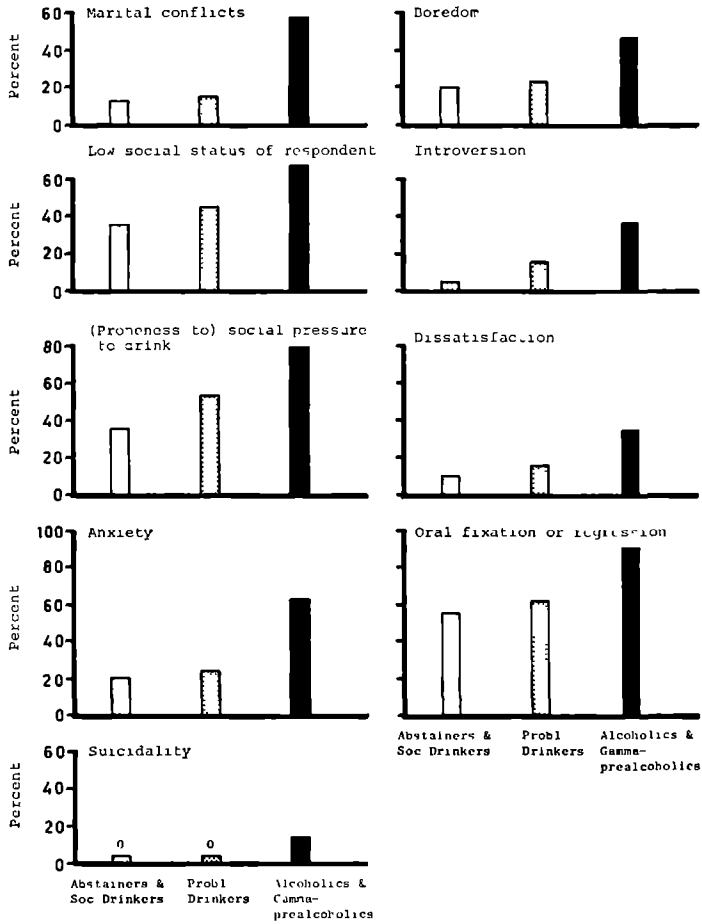


figure 59

All respondents perpetuating factors.

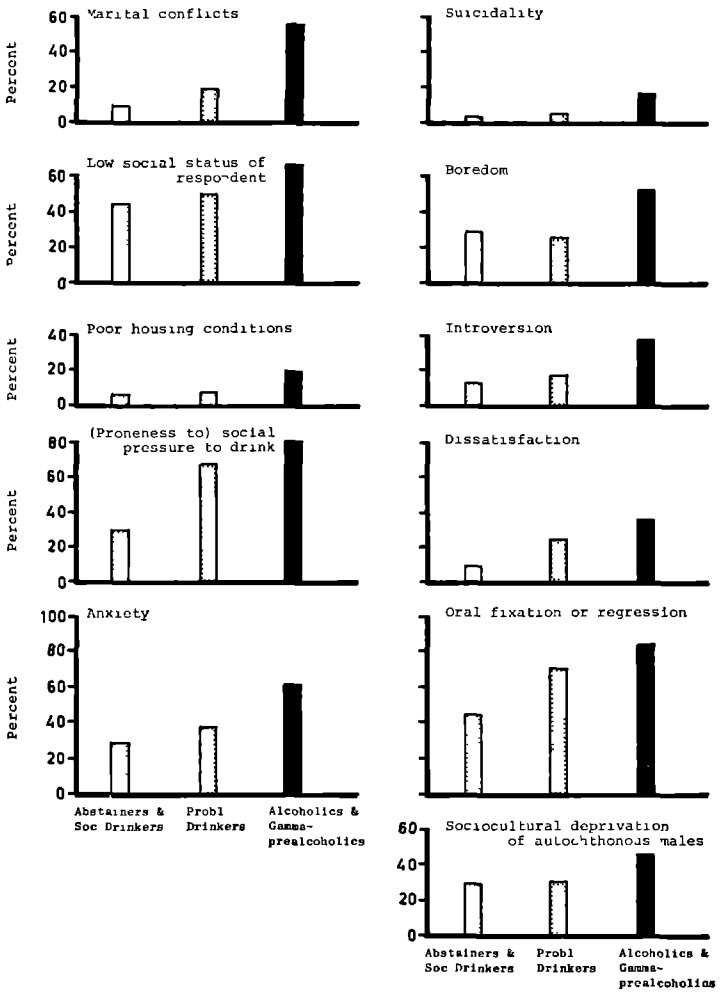


Table 5 19

Perpetuating factors in survey A Mean factor scores (± S E M) per diagnostic category

Factor	Diagnostic category				R	p
	Abstinence (N = 181)	Social Drinking (N = 371)	Problem Drinking (N = 83)	Prealcoholism & Gamma-prealcoholism & Alcoholism (N = 73)		
Social normativity	525 ± 7	504 ± 5	462 ± 11	454 ± 14	—0 1208	0 001
Excessive smoking	453 ± 5	497 ± 5	543 ± 12	580 ± 13	0 3669	0 001
Excessive sweets consumption	510 ± 8	502 ± 5	500 ± 12	464 ± 10	—0 1208	0 001

R = Pearson correlation coefficient
p = Level of significance

Table 5 20

Perpetuating factors in percent per diagnostic category in survey A

Factor	Diagnostic category				χ^2	df	p
	Abstinence (N = 181)	Social Drinking (N = 371)	Problem Drinking (N = 83)	Prealcoholism & Gamma prealcoholism & Alcoholism (N = 73)			
(Proneness to) social pressure	11	40	45	73	95 90	3	0 000
Hospitality drinking[1]	47	48	42	59	3 74	3	n s
Drugs	3	2	6	7	7 24	3	0 06

[1] 123 abstainers, 274 social drinkers, 65 problem drinkers, and 58 alcoholics & (gamma-)prealcoholics

5.4. SUMMARY AND CONCLUSIONS

A total of *12 predisposing factors* in any of the subdivisions evaluated in survey B & C was found to correlate significantly with the alcoholismic gradient:

— paternal inebriety in one's youth;

— interparental conflicts in one's youth;
— parent-child conflicts in one's youth;
— P.B.I.-2
— P.B.I.-3
— P.B.I.-4
— inebriety in extended family;
— "genetic" autochthony;
— rural domicile;
— low educational level;
— male sex;
— economic availability (of alcohol).

These factors are shown per subdivision in *table 6.1.*

A total of *13 perpetuating factors* in any of the subdivisions evaluated in survey B & C was found to correlate significantly with the alcoholismic gradient:
— marital conflicts;
— low social status of respondent;
— poor housing conditions;
— violation of one's confidence by relatives and significant others;
— (proneness to) social pressure to drink;
— sociocultural deprivation of autochthonous males;
— anxiety;
— suicidality;
— boredom;
— introversion;
— dissatisfaction;
— oral fixation or regression;
— drugs.

These factors are shown per subdivision in *table 6.1.*

Hence it is evident that about *50%* of the factors evaluated were not considered as significant. This percentage definitely *exceeds* 50% among female respondents.

Non-significant correlations found in this study can be:
(1) truly non-significant correlations;
(2) spuriously non-significant correlations, i.e. caused by the fact that a specific factor's *expected* prevalence is too low among all

diagnostic categories evaluated; this was especially true among female respondents, however, understandably because of the very small group of females.

The 12 predisposing and 13 perpetuating factors will be conjointly evaluated in Chapter 6.

ANALYTICAL EPIDEMIOLOGY: CONJOINT EVALUATION OF PREDISPOSING AND PERPETUATING FACTORS IN ALCOHOLISM AND ITS PRECURSOR STAGES

6.1. INTRODUCTION

Predisposing and perpetuating factors can be considered as *handicaps* or *risk factors* predisposing to or perpetuating alcoholism.

6.2. METHODS

For the conjoint evaluation of these *handicaps* or *risk factors* a "*Risk Factor Analysis*" (*R.F.A.*) is applied, implying a *summation* of those predisposing and perpetuating factors significantly correlated with the alcoholismic gradient. In this *R.F.A.* the *mean* number of predisposing or perpetuating factors is determined per diagnostic category (Cahalan & Cisin, 1976; Salel et al., 1977; Wallace, 1972).

For this conjoint evaluation also another method is applied, i.e. *discriminant function analysis* (*D.F.A.*).

Table 6.1. shows the subdivisions and the factors participating in the *R.F.A.* and the *D.F.A.*

Furthermore, to assess the hypothesis that the classification in diagnostic categories - as conducted in Chapter 2 - implies a truthful alcoholismic gradient, *mean* factor scores for the factor-analytical factor "problem drinking" (cf. Chapter 2) were determined (plus range) for these diagnostic categories in survey B & C. As shown in *fig. 6.1.*, this hypothesis is convincingly confirmed. Factor scores for this factor range between 367 and 666.

The *R.F.A.* and *D.F.A.* are applied in survey B & C:

(1) for predisposing factors *separately;*

(2) for the *combination* of predisposing and perpetuating factors.

figure 6 1

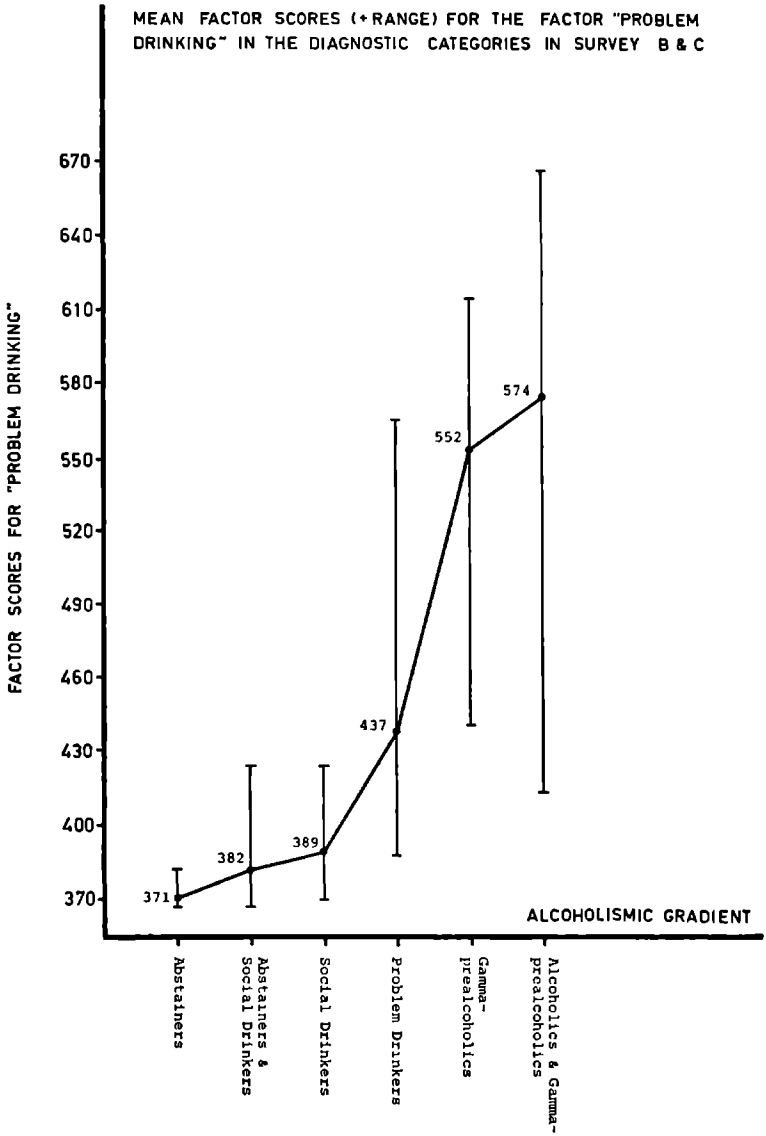

MEAN FACTOR SCORES (+RANGE) FOR THE FACTOR "PROBLEM DRINKING" IN THE DIAGNOSTIC CATEGORIES IN SURVEY B & C
FACTOR SCORES FOR "PROBLEM DRINKING"
670
640
610
580
550
520
490
460
430
400
370
371
382
389
437
552
574
ALCOHOLISMIC GRADIENT
Abstainers
Abstainers & Social Drinkers
Social Drinkers
Problem Drinkers
Gamma-prealcoholics
Alcoholics & Gamma-prealcoholics

Table 6.1

Predisposing and perpetuating factors and subdivisions employed in the conjoint evaluation of the risk factor analysis (R F A) and the discriminant function analysis (D F A)

Females	*Males <30 years*	*Males >30 years*	*All respondents*
a) Predisposing factors	a) Predisposing factors	a) Predisposing factors	a) Predisposing factors
1) Paternal inebriety	1) Paternal inebriety	1) Paternal inebriety	1) Paternal inebriety
2) Inebriety in extended family	2) Interparental conflicts	2) P B I -3	2) Interparental conflicts
3) Low educational level	3) Parent child conflicts	3) P B I -4	3) Inebriety in extended family
	4) P B I -2	4) Inebriety in extended family	4) 'Genetic" autochthony
	5) P B I -4	5) "Genetic" autochthony	5) Rural domicile
	6) Inebriety in extended family	6) Rural domicile	6) Low educational level
	7) Rural domicile	7) Low educational level	7) Male sex
	8) Low educational level		8) Economic availability
	9) Economic availability		
b) Perpetuating factors	b) Perpetuating factors	b) Perpetuating factors	b) Perpetuating factors
1) Marital conflicts	1) Marital conflicts	1) Marital conflicts	1) Marital conflicts
2) Poor housing conditions	2) Violation of confidence	2) Low social status of respondent	2) Low social status of respondent
3) Violation of confidence	3) (Proneness to) social pressure	3) (Proneness to) social pressure	3) Poor housing conditions
4) (Proneness to) social pressure	4) Sociocultural deprivation Aruban males	4) Anxiety	4) (Proneness to) social pressure
5) Suicidality	5) Anxiety	5) Suicidality	5) Sociocultural deprivation Aruban males
	6) Boredom	6) Boredom	6) Anxiety
	7) Introversion	7) Introversion	7) Suicidality
	8) Dissatisfaction	8) Dissatisfaction	8) Boredom
	9) Oral fixation or regression	9) Oral fixation or regression	9) Introversion
	10) Drugs		10) Dissatisfaction
			11) Oral fixation or regression

The *R.F.A.* and *D.F.A.* are applied among the following subdivisions:

(1) female respondents;
(2) male respondents younger than 30 years *without* P.B.I.-factors;
(3) male respondents younger than 30 years *with* P.B.I.-factors;
(4) male respondents older than 30 years *without* P.B.I.-factors;
(5) male respondents older than 30 years *with* P.B.I.-factors;
(6) all female and male respondents *without* P.B.I.-factors.

Analogous types of conjoint evaluation have been conducted by other authors (Bell et al., 1976; Cahalan & Cisin, 1976; Wallace, 1972).

In Bell's study, however, this only implied a summation of "stressful life events" but not other factors facilitating the alcoholismic gradient, like in this study and in those of Wallace and Cahalan et al. (Bell et al., 1976; Cahalan & Cisin, 1976; Wallace, 1972).

It should be emphasized, that the R.F.A. - by its intrinsic nature of being a mere summation of risk factors - precludes an exact evaluation of the possible differences in *weight* per (predisposing or perpetuating) factor. A multiple regression analysis could have overcome this difficulty.

However, since the vast majority of predisposing and perpetuating factors in this study - with the exception of the P.B.I.-factors - are not of a true ordinal nature, a multiple regression analysis could not be applied.

6.3. RESULTS WITH THE RISK FACTOR ANALYSIS (R.F.A.)

The results of the conjoint evaluation with the *R.F.A.* are shown below separately for each subdivision.

Each figure showing these results, presents the *mean* number of predisposing or predisposing plus perpetuating factors ± S.E.M. per diagnostic category, and R (Kendall's Tau C).

6.3.1. R.F.A. results among female respondents

These are shown in *fig. 6.2.* and *tables 6.2. & 6.3.*

6.3.2. R.F.A. results among male respondents younger than 30 years without P.B.I.-factors

These are shown in *fig. 6.3.* and *tables 6.2. & 6.3.*

6.3.3. R.F.A. results among male respondents younger than 30 years with P.B.I.-factors

These are shown in *fig. 6.4.* and *tables 6.2. & 6.3.*

6.3.4. R.F.A. results among male respondents older than 30 years without P.B.I.-factors

These are shown in *fig. 6.5.* and *tables 6.2. & 6.3.*

Table 6.2.

Risk factor analysis (R.F.A.). Predisposing factors in survey B & C.

Subdivision	Mean number of predisposing factors (± S.E.M.)	R	p
Female respondents		0.60111	0.0003
Abstainers & Social Drinkers	0.58 ± 0.17		
Problem Drinkers & (Gamma-pre-) alcoholics	1.64 ± 0.20		
Male respondents younger than 30 years, without P.B.I.-factors		0.56560	0.0000
Abstainers & Social Drinkers	1.12 ± 0.21		
Problem Drinkers	2.67 ± 0.47		
Alcoholics & Gamma-prealcoholics	3.80 ± 0.61		
Male respondents younger than 30 years, with P.B.I.-factors		0.43526	0.0013
Abstainers & Social Drinkers	1.77 ± 0.28		
Problem Drinkers	3.71 ± 0.68		
Alcoholics & Gamma-prealcoholics	4.50 ± 1.56		
Male respondents older than 30 years, without P.B.I.-factors		0.25248	0.0000
Abstainers & Social Drinkers	1.05 ± 0.24		
Problem Drinkers	2.15 ± 0.45		
Alcoholics & Gamma-prealcoholics	2.58 ± 0.12		
Male respondents older than 30 years, with P.B.I.-factors		0.38699	0.0000
Abstainers & Social Drinkers	2.00 ± 0.28		
Problem Drinkers	2.75 ± 0.37		
Alcoholics & Gamma-prealcoholics	3.88 ± 0.21		
All respondents (without P.B I.-factors)		0.40430	0.0000
Abstainers & Social Drinkers	2.54 ± 0.19		
Problem Drinkers	3.71 ± 0.30		
Alcoholics & Gamma-prealcoholics	4.43 ± 0.14		

The *numbers* (N) are shown in *tables 5.7., 5.8. & 5.9.* (Chapter 5).

6.3.5. R.F.A. results among male respondents older than 30 years with P.B.I.-factors

These are shown in *fig. 6.6.* and *tables 6.2. & 6.3.*

6.3.6. R.F.A. results among all female and male respondents without P.B.I.-factors

These are shown in *fig. 6 7* and *tables 6.2. & 6 3*

Table 6 3

Risk factor analysis (R F A) Predisposing plus perpetuating factors in survey B & C

Subdivision	Mean number of predisposing plus perpetuating factors (± SEM)	R	p
Female respondents		0 83379	0 0000
Abstainers & Social Drinkers	1 33 ± 0 22		
Problem Drinkers & (Gamma-pre-) alcoholics	4 29 ± 0 37		
Male respondents younger than 30 years, without P B I -factors		0 70506	0 0000
Abstainers & Social Drinkers	2 76 ± 0 31		
Problem Drinkers	7 22 ± 0 89		
Alcoholics & Gamma-prealcoholics	9 40 ± 1 02		
Male respondents younger than 30 years, with P B I -factors		0 54545	0 0001
Abstainers & Social Drinkers	3 41 ± 0 38		
Problem Drinkers	8 29 ± 1 23		
Alcoholics & Gamma-prealcoholics	9 00 ± 2 27		
Male respondents older than 30 years, without P B I -factors		0 39724	0 0000
Abstainers & Social Drinkers	3 00 ± 0 47		
Problem Drinkers	4 69 ± 0 75		
Alcoholics & Gamma-prealcoholics	7 35 ± 0 22		
Male respondents older than 30 years, with P B I -factors		0 53398	0 0000
Abstainers & Social Drinkers	4 18 ± 0 50		
Problem Drinkers	4 63 ± 0 50		
Alcoholics & Gamma-prealcoholics	8 88 ± 0 34		
All respondents (without P B I -factors)		0 58558	0 0000
Abstainers & Social Drinkers	4 80 ± 0 28		
Problem Drinkers	7 17 ± 0 55		
Alcoholics & Gamma-prealcoholis	9 75 ± 0 24		

The numbers (N) are shown in *tables 5 16 , 5 17 & 5 18* (Chapter 5)

figure 6.2.

R.F.A.-results among female respondents.

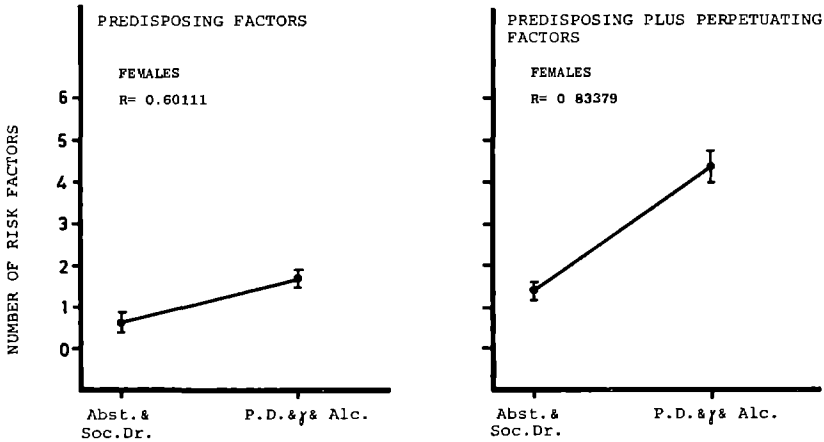


figure 6.3.

R.F.A.-results among male respondents younger than 30 years, without P.B.I.-factors.

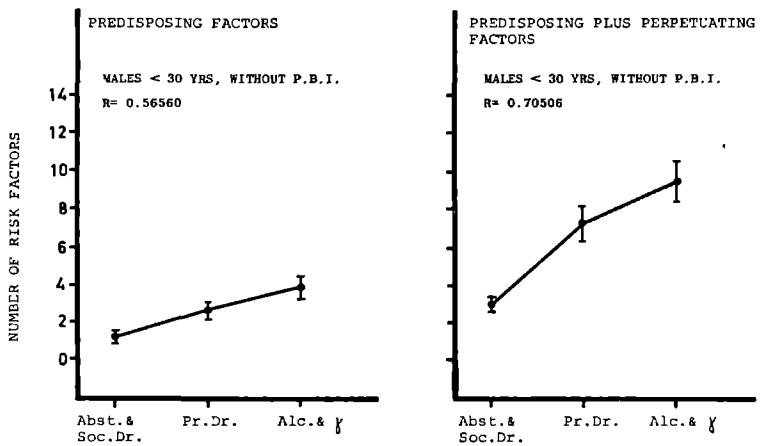


figure 6.4.

R.F.A.-results among male respondents younger than 30 years, with P.B.I.-factors.

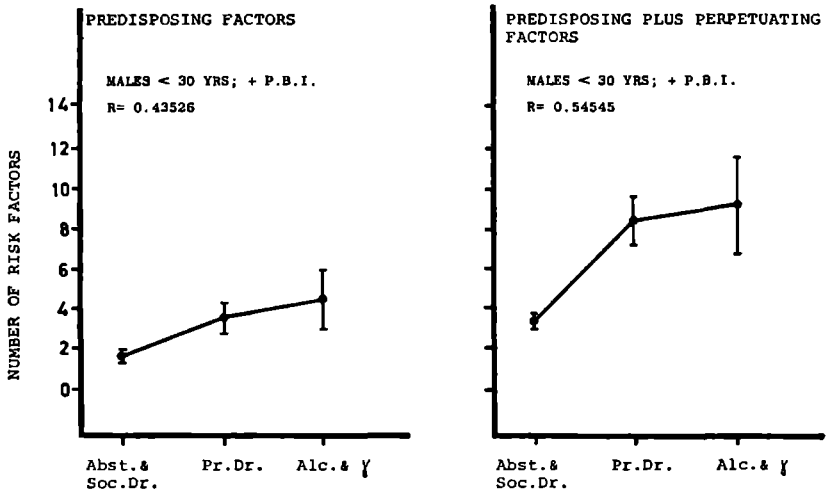


figure 6.5.

R.F.A.-results among male respondents older than 30 years, without P.B.I.-factors.

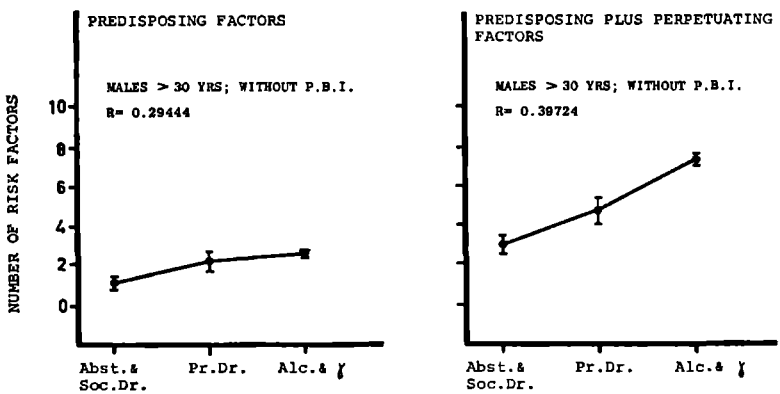


figure 6 6

R.F.A.-results among male respondents older than 30 years, with P.B.I.-factors.

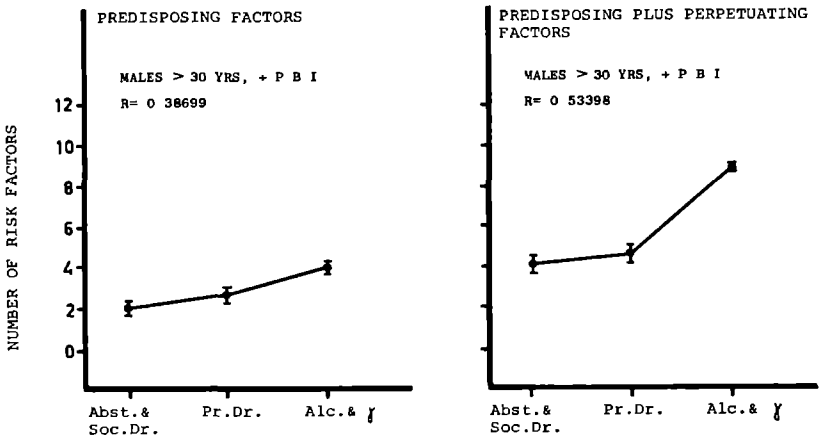
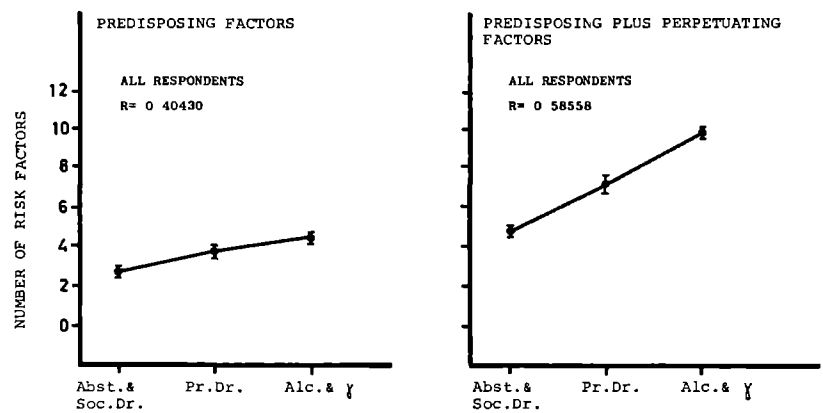


figure 6 7.

R.F.A.-results among all female and male respondents, without P.B.I.-factors.



6.3.7. Discussion of results with the R.F.A.

As shown in *tables 6.2. and 6.3.* and in *fig. 6.2. through 6.7.*, the addition of the perpetuating factors to the predisposing factors in the R.F.A. gives rise to *different* results in the six subdivisions studied, i.e. when the various correlation coefficients (R) are compared with each other. Among *male* respondents these differences in results are ascribable to the effect of *age:* the increase of the correlation coefficient (R) among *males younger than 30 years* - both *with* and *without* P.B.I.-factors - when the perpetuating factors are added to the predisposing factors, is *less* than in the *four* remaining categories, though not impressively. The following conclusions can be drawn:

(1) *Under the age of 30 years among male respondents the predisposing factors show a relatively stronger correlation with the alcoholismic gradient* than over that age;

(2) *Over the age of 30 years among male respondents the perpetuating factors show a relatively stronger correlation with the alcoholismic gradient* than under that age.

The reason for this difference is not entirely clear, though it may be presumed that at *younger* ages (e.g. under 30 years) most of the predisposing factors occurring in youth will have greater influence on the alcoholismic gradient because the time lapse (between the occurrence of these predisposing factors and the development of problem drinking or alcoholism) is *shorter* than at older ages (e.g. over 30 years).

Over 30 years evidently there is a relative coincidence in time between most of the perpetuating factors and the development of problem drinking and alcoholism, hence above that age the latter factors will have a greater influence on the alcoholismic gradient. In other words: under 30 years most of the predisposing factors are fresher in the individual's memory than over that age, while over 30 years most of the perpetuating factors are better memorized.

This finding supports the usefulness of a subdivision into predisposing and perpetuating factors.

For *female respondents* this effect of *age* could not be adequately evaluated, since the very small number of females did not permit a subdivision in categories under and over 30 years. However, among

female respondents the addition of perpetuating factors to the predisposing factors also gives rise to an increase of the correlation coefficient (R) even more impressive than among male respondents *older* than 30 years. This is understandable, since among female respondents alcoholics (& gamma-prealcoholics & problem drinkers) *over* 30 years constitute 86% of all females in these diagnostic categories.

Moreover, it has been reported that female alcoholism starts at a *later age* as compared to male alcoholism (Cadoret, 1976; Dahlgren, 1975; Gomberg, 1976; Holzgreve, 1970; Lolli, 1953), and that such factors as marital conflicts, divorce, sexual disturbances, prostitution, depression, suicidality, negative life expection, unsatisfactory living conditions - all of which may be considered perpetuating factors - play important roles in the pathogenesis of female alcoholism (Dahlgren, 1975; Rieth, 1970). However, research on female alcoholism has not yet reached the extent of research on male alcoholism, and hence any conclusion in this section should be *tentative* (Dahlgren, 1975; Rieth, 1970).

Alcoholism in females may develop relatively quickly provoked by some sudden or recent stressful life event (Lolli, 1953).

Prostitution was found among *25%* of female alcoholics in this study, and sexual promiscuity in another *17%* (data derived from patients' records). Sexual disturbances in females are considered to be a primary predisposing factor and not a result of alcoholism (Dahlgren, 1975). The higher correlation coefficients (R) among female respondents as compared to male respondents in this study, are in agreement with the statement that female alcoholism is more rooted in external (social, environmental) factors than male alcoholism (Dahlgren, 1975).

For *all female and male respondents* the addition of perpetuating factors to the predisposing factors gives rise to an increase of the correlation coefficient (R) comparable to that among male respondents *older* than 30 years. This is understandable, since in this subdivision (all respondents) the majority consists of males older than 30 years.

As shown in *tables 6.2. & 6.3.* and *figs. 6.2. through 6.7.* all the predisposing and perpetuating factors conjointly evaluated are signi-

ficantly more correlated with the alcoholismic gradient among *male respondents younger than 30 years* as compared to males older than 30 years. This is in agreement with Cahalan's findings (Cahalan & Cisin, 1976). Thus, while the highest rate of predisposing and perpetuating factors are found among (gamma-pre-)alcoholics and problem drinkers as compared to abstainers and social drinkers in males *under* the age of 30, this finding is in contrast with the observation that the *prevalence* of male (and female) *alcoholism* is higher *above* 30 years.

These age-trend findings suggest that many *male* alcoholics (average age 45 years) have formed and maintained their excessive drinking habits on the basis of predisposing and perpetuating factors to a large extent existing before the age of 30 years.

The traditional expectation concerning alcoholism is that alcoholics accumulate increasing numbers of predisposing and perpetuating factors throughout their lives (Cahalan & Cisin, 1976).

The findings in this study (among male respondents) are at variance with this expectation, as described above.

In Cahalan's longitudinal study it was found, that continuity of *specific* problems (i.e. perpetuating factors) over time is low, but that continuation of *some* alcohol problems (i.e. problem drinking, as defined in this study) is very likely for those who already were problem drinkers at the start of that study (Cahalan & Cisin, 1976). Situational factors (i.e. perpetuating factors) were considered to have great influence in determining whether the individual would tend to continue being a problem drinker (Cahalan & Cisin, 1976). Cahalan's observations are suggestive for *predominance* of the shift: perpetuating factors → alcoholism as compared to the shift in the opposite direction. A study of the impact of *events* (such as perpetuating and predisposing factors) on changes in drinking behavior is being conducted by Cahalan et al., to be reported upon in the years ahead (Cahalan & Cisin, 1976).

Of course, only follow-up (longitudinal) studies in the same group of respondents would confirm or reject any predominant direction in the circular causality relationship between alcoholism and its perpetuating factors.

6.4. RESULTS WITH THE DISCRIMINANT FUNCTION ANALYSIS (D.F.A.)

The results of the conjoint evaluation with the *discriminant function analysis* are shown below separately for each subdivision in *tables 6.4., 6.5. and 6.6.*

Because application of the discriminant function analysis is more difficult when dealing with $>$ 2 categories, in this study a subdivision in *two* groups is used, i.e.:

Group 1: Abstainers & Social Drinkers;

Group 2: Problem Drinkers & Gamma-prealcoholics & Alcoholics.

Table 6.4.

Discriminant function analysis. Pred'sposing factors in survey B & C.

Actual group per subdivision	N	Predicted group membership in percent: Group 1	Group 2	Percent of "grouped" cases correctly classified
Female respondents				76
Group 1	24	71	29	
Group 2	14	14	86	
Male respondents younger than 30 years, without P.B.I.-factors				84
Group 1	25	84	16	
Group 2	19	16	84	
Male respondents younger than 30 years, with P.B.I.-factors				82
Group 1	22	91	9	
Group 2	11	18	82	
Male respondents older than 30 years, without P.B.I.-factors				73
Group 1	20	90	10	
Group 2	122	30	70	
Male respondents older than 30 years, with P.B.I.-factors				77
Group 1	17	88	12	
Group 2	68	26	74	
All respondents (without P.B.I.-factors)				74
Group 1	69	84	16	
Group 2	155	30	70	

It is evident that this implies a dichotomization in "pathological drinking" versus "absence of pathological drinking".

In *tables 6.4. and 6.5. all* predisposing or predisposing *plus* perpetuating factors are conjointly evaluated.

Table 6 5

Discriminant function analysis. Predisposing plus perpetuating factors in survey B & C

Actual group per subdivision	N	Predicted group membership in percent: Group 1	Group 2	Percent of "grouped" cases correctly classified
Female respondents				90
Group 1	24	88	12	
Group 2	14	7	93	
Male respondents younger than 30 years, without P B I -factors				93
Group 1	25	96	4	
Group 2	19	10	90	
Male respondents younger than 30 years, with P B I -factors				100
Group 1	22	100	0	
Group 2	11	0	100	
Male respondents older than 30 years, without P B I -factors				82
Group 1	20	80	20	
Group 2	122	18	82	
Male respondents older than 30 years, with P B I -factors				84
Group 1	17	88	12	
Group 2	68	18	82	
All respondents (without P B I -factors)				85
Group 1	69	87	13	
Group 2	155	15	85	

In *table 6.6. "stepwise" discriminant function analysis* is shown. This implies that only those risk factors with the greatest discriminating capacity are selected step by step, i.e. in rank order of their degrees of correlation with the alcoholismic gradient. Moreover in this procedure the predisposing factors are first evaluated, followed by the perpetuating factors.

Table 6.6.

"Stepwise" discriminant function analysis. Predisposing plus perpetuating factors in survey B & C.

Actual group per subdivision	N	Predicted group membership in percent: Group 1	Group 2	Percent of "grouped" cases correctly classified
Female respondents				90
Group 1	24	92	8	
Group 2	14	14	86	
Male respondents younger than 30 years, without P.B.I.-factors				93
Group 1	25	96	4	
Group 2	19	10	90	
Male respondents younger than 30 years, with P.B.I.-factors				94
Group 1	22	96	4	
Group 2	11	9	91	
Male respondents older than 30 years, without P.B.I.-factors				80
Group 1	20	85	15	
Group 2	122	20	80	
Male respondents older than 30 years, with P.B.I.-factors				82
Group 1	17	88	12	
Group 2	68	19	81	
All respondents (without P.B.I.-factors)				83
Group 1	69	87	13	
Group 2	155	19	81	

With the "stepwise" discriminant function analysis (cf. *table 6.6.*) a *smaller* number of risk factors (as compared to those in *table 6.1.*) is considered as significant. These significant factors are shown in *table 6.7.*

The findings in *table 6.7.* reflect, that "stepwise" discriminant function analysis selects only the most significant factors.

The findings in *table 6.7.* also indicate, that among all subdivisions studied *paternal inebriety* is the most significant predisposing factor; this supports the view, that adequate treatment of male alcoholism implies adequate prevention of alcoholism - of course, amongst other preventive measures.

Table 6.7.

Predisposing and perpetuating factors found significant with the "stepwise" discriminant function analysis.

Subdivision	Predisposing factors	Perpetuating factors
Female respondents	1) Paternal inebriety 2) Low educational level	1) Marital conflicts 2) Poor housing conditions 3) Violation of confidence 4) (Proneness to) social pressure 5) Suicidality
Males younger than 30 years, without P.B.I.-factors	1) Paternal inebriety 2) Economic availability 3) Parent-child conflicts 4) Rural domicile 5) Interparental conflicts	1) Marital conflicts 2) Violation of confidence 3) (Proneness to) social pressure 4) Sociocultural deprivation Aruban males 5) Oral fixation or regression 6) Drugs
Males younger than 30 years, with P.B.I.-factors	1) Paternal inebriety 2) P.B.I.-2 3) Economic availability 4) Inebriety in extended family 5) P.B.I.-4	1) Violation of confidence 2) Sociocultural deprivation Aruban males 3) Oral fixation or regression
Males older than 30 years, without P.B.I.-factors	1) Paternal inebriety 2) "Genetic" autochthony 3) Inebriety in extended family	1) Marital conflicts 2) (Proneness to) social pressure 3) Anxiety 4) Introversion 5) Dissatisfaction 6) Oral fixation or regression 7) Low social status of respondent
Males older than 30 years, with P.B.I.-factors	1) Paternal inebriety 2) P.B.I.-3 3) "Genetic" autochthony	1) (Proneness to) social pressure 2) Suicidality 3) Anxiety 4) Introversion 5) Oral fixation or regression

All respondents (without P B I -factors)	1) Paternal inebriety	1) Marital conflicts
	2) Male sex	2) Poor housing conditions
	3) Low educational level	3) (Proneness to) social pressure
	4) Inebriety in extended family	4) Suicidality
	5) Economic availability	5) Anxiety
		6) Boredom
		7) Introversion
		8) Dissatisfaction
		9) Oral fixation or regression

6.4.1. Discussion of results with the D.F.A.

As shown in *table 6.4., 6.5. & 6.6.*, the *risk factors* (predisposing and perpetuating factors) studied have a substantial predicting capacity. This is of great importance, since it implies that for example in general practice it is possible to detect those individuals at high risk of progression on the alcoholismic gradient (i.e. alcoholics, (gamma-) prealcoholics and even problem drinkers) by simply asking the questions reflecting these risk factors when writing down a patient's medical history (Hore & Wilkins, 1976; Wilkins, 1973, 1975, 1976, 1976a, 1977). Thus having detected the patient at high risk of developing alcoholism, one can proceed by asking the questions considered as "diagnostic" for alcoholism and its precursor stages, as mentioned in Chapter 2.

In a study in Manchester a $\geqslant$ 50% probability of detecting alcoholism was found correlated with the following (risk) factors: admitting a previous drunkenness offence; having sought help previously for drinking problems; smell of alcohol at consultation; gastritis or peptic ulcer; previous accidents at work or on the road; having requested a sick note for symptoms which did not appear genuine; inebriety in near relatives (Hore & Wilkins, 1976; Wilkins, 1977).

Some other important risk factors mentioned in the Manchester study are: low social status; single males over 40 years; divorce; marital problems; poor housing conditions; ethnic group; anxiety; depression; attempted suicide; insomnia; paranoia; violent outbursts; hallucinations; unemployment; repeated changes of employment; heavy cigarette smoking; taking drugs (Wilkins, 1973, 1975a,

1976a). Many of the risk factors included in the present study were also evaluated by Wilkins, as mentioned above.

It should be mentioned that for both "predisposing factors" and the combination of "predisposing plus perpetuating factors" (cf. *tables 6.4., 6.5. & 6.6.)* among the subdivision "all respondents" six aselect samples were taken (approx. 80% of respondents); in all these samples approximately the same percentages of "grouped" cases correctly classified were found as in the total of 224 respondents.

The findings in *table 6.6.* indicate that with a *smaller number of risk factors* almost *the same predicting capacity* is obtained as compared to a conjoint evaluation of all factors mentioned in *table 6.1.* and evaluated in *table 6.5.*

Among *female respondents* there is a substantial increase in predicting capacity when the perpetuating factors are added to the predisposing factors; this is in agreement with the R.F.A. findings.

Among *male respondents younger than 30 years* (with P.B.I.-factors) however, this increase is most impressive. This is at variance with the R.F.A. findings, where it was shown that perpetuating factors play a more significant role above 30 years among males.

Among *male respondents younger than 30 years* (with or without P.B.I.-factors) the predicting capacity of the predisposing factors (*table 6.4.*) and of the predisposing *plus* perpetuating factors (*tables 6.5. & 6.6.)* is better than among the remaining subdivisions; this is in agreement with the R.F.A. findings where the predisposing factors are concerned.

Table 6.7. shows the predisposing and perpetuating factors found significant with the "stepwise" discriminant function analysis in the six subdivisions studied; the number before every factor reflects the "step" at which this factor enters the D.F.A. The sequence of the "steps" in *table 6.6.* implies "stepwise" summation of those factors conjointly possessing the greatest discriminating capacity between "Group 1" and "Group 2".

A comparison of *tables 6.1. and 6.7.* reveals which of the predisposing and perpetuating factors "dropped out" i.e. were found not significant with the "stepwise" D.F.A. Factors which "dropped out" in the "stepwise" D.F.A. did so, because of their significant intercorrelations with factors preceding them in this procedure.

In the six subdivisions studied there appeared to be *no* apparent pattern for these intercorrelations. Therefore is was not possible to describe or characterize these clusters of intercorrelations for any of the six subdivisions.

The results with the "stepwise" D.F.A. as shown in *table 6.6.* indicate, however, that when repeating this study one could consider to employ a smaller number of predisposing and perpetuating factors for each subdivision, as shown in *table 6.7.*

6.5. ALCOHOLISM AND EXCESSIVE ALCOHOL CONSUMPTION (I.E. THE ALCOHOLISMIC GRADIENT) AS RISK FACTOR TO OTHER DISEASES

Alcoholism and its precursor stages imply a separate *risk factor* to many other diseases (Criteria Committee, 1972; De Lint, 1975a; De Lint & Schmidt, 1970a; Eichner, 1973; Erkelens, 1975, 1976; Jellinek, 1942; Klatsky et al., 1977; Lieber, 1973, 1973a; Liu, 1973; Roos, 1976; Schmidt & De Lint, 1972; Smith & Lucie, 1973; Spodick et al., 1972; Que, 1975).

A great number of these diseases are mentioned in Chapter 7.

The *risk of death* from various causes increases with increasing alcohol consumption (De Lint, 1975a). Deaths observed in samples of excessive drinkers were attributable to causes such as: liver cirrhosis; severe alcohol intoxication; cardiovascular diseases (due to stimulation of arteriosclerosis and hypertension, and excessive smoking); alcoholic cardiomyopathy; tuberculosis and other bacterial pneumonia (due to susceptibility to infection); bronchial carcinoma (due to the alcoholic's excessive smoking); cancers of the upper digestive tract; suicides; traffic and other accidents (De Lint, 1975a; Schmidt & De Lint, 1972).

The fetal alcohol syndrome is also acknowledge to be caused during pregnancy by alcoholism of the mother (Gomberg, 1976; Jones et al., 1976; Ouelette & Rosett, 1976; Pytkowicz Streissguth, 1976; Roos, 1976; Rosett, 1976; Rosett et al., 1976; Smith et al., 1976).

Apart from liver damage (alcoholic steatosis, hepatitis, cirrhosis) lesions in the striated muscles (alcoholic myopathy and cardiomyopathy), pancreatitis, gastritis, peptic ulcer, gastro-enteritis, anemia, leukopenia, thrombocytopenia, metabolic lactic acidosis, cardiovas-

cular diseases, hypertension, arteriosclerosis, bacterial pneumonia, tuberculosis, bronchial carcinoma, upper digestive tract malignancies, hyperuricemia, gout, hypertriglyceridemia, beriberi, polyneuropathy, Wernicke syndrome and Korsakoff syndrome are the best known somatic complications of alcoholism.

It is evident that prevention of (deaths due to) these alcohol-related diseases implies prevention and treatment of alcoholism. However, adequate prevention and treatment of alcoholism (and hence of alcohol-related health damage) implies appropriate measures to prevent and eliminate the *risk factors* (i.e. predisposing and perpetuating factors) conducive to and perpetuating *alcoholism.* This is the perspective, in which the *handicaps* (i.e. risk factors; predisposing and perpetuating factors) discussed in this study have to be viewed.

6.6. CONCLUSIONS

1) In all of the six subdivisions studied an increase on the alcoholismic gradient (abstinence & social drinking → problem drinking → (gamma)-prealcoholism & alcoholism) is correlated with increases in mean number of *handicaps* (i.e. *risk factors; predisposing* and *perpetuating factors).* This would suggest that the development of alcoholism and its precursor stages is positively stimulated and perpetuated by increasing numbers of these factors (R.F.A. findings). Analogous results have been found in studies of risk factors in coronary heart disease (Arntzenius et al., 1976; Arntzenius & Styblo, 1977; De Haas, 1976, 1977; Sluyter et al., 1977; Styblo et al., 1977; Salel et al., 1977).
2) Under the age of 30 years among male respondents *predisposing factors* probably play a relatively more important role than over 30 years in the development of alcoholism (R.F.A. and D.F.A. findings).
3) Over the age of 30 years among male respondents *perpetuating factors* probably play a relatively more important role than under 30 years in the development of alcoholism (R.F.A. findings).
4) Among female respondents of all ages *perpetuating factors* probably play a relatively more important role than among males younger than 30 years (R.F.A. and D.F.A. findings).
5) The *risk factors* or *handicaps* (i.e. *predisposing and perpetuating*

factors) evaluated in this study are not only conducive to alcoholism, but indirectly also to a great number of diseases caused by alcoholism. Some alcohol-related diseases are mentioned in Chapter 7.

6) Discriminant function analysis reveals a substantial predicting capacity of the predisposing and perpetuating factors evaluated, ranging between 70% and 100%.
7) "Stepwise" discriminant function analysis reveals that with a smaller number of risk factors almost the same predicting capacity is obtained as compared to a conjoint evaluation of all risk factors mentioned in *table 6.1.*
8) The data obtained through R.F.A. and D.F.A. can be briefly described as *risk profiles* for the alcoholismic gradient.
9) Sensitivity and specificity of the conjoint evaluation of all risk factors with the D.F.A. range between 70% and 100%, which are moderately high values.

Chapter 7

OPERATIONAL RESEARCH : THERAPEUTIC REGIMENS AND RESULTS; FACTORS AFFECTING THERAPEUTIC RESULTS

7.1. PATIENTS AND DIAGNOSES

From October 1969 through September 1972, 248 patients were either hospitalized or treated at the alcoholism outpatient clinic, under the diagnoses :

1) alcohol addiction: a) gamma-alcoholism;
   b) delta-alcoholism;
   c) chronic alcoholism;
2) gamma-prealcoholism;
3) problem drinking: a) Jellinek's alpha-alcoholism;
   b) Jellinek's beta-alcoholism;
   c) Jellinek's epsilon-alcoholism.

It should be mentioned, that only gamma-alcoholism, delta-alcoholism and chronic alcoholism imply true addiction, while the other diagnoses indicate precursor stages. In that period (October 1969 through September 1972) an average of 8-10 alcoholics were hospitalized monthly with an average stay of three weeks. Among these patients about *80* alcoholismic diagnoses were established, i.e. diagnoses of ailments caused by either withdrawal or intoxication, or of deviations conducive to excessive drinking (cf. *table 7.1.)*. Moreover, about *25* other psychiatric diagnoses were established (cf. *table 7.2.)*, as well as about *10* neurological diagnoses (cf. *table 7.2.)*, *30* internal medical diagnoses, *6* dermatological diagnoses and *3* surgical diagnoses.

Table 7.1.
Alcoholismic Diagnoses.

1) *Syndromes and symptoms due to alcohol withdrawal*
   Alcoholic hangover
   Alcoholic tremulousness
   Alcohol withdrawal syndrome
   Acute alcoholic hallucinosis
   Alcoholic convulsions
   Alcoholic hypomagnesemia
   Blackout

Delirium tremens
Extrapyramidal syndrome in delirium tremens
Transient hypertension in alcohol withdrawal syndrome
Alcoholic craving

2) *Syndromes and symptoms due to acute intoxication*
   a) *Psychiatric*
      Alcoholic depression
      Acute alcoholic intoxication
      Heilbronner's pathological intoxication
      Alcoholic aggressive and psychopathiform behavior
      Alcoholic psychosis
      Disulfiram-induced acetaldehyde psychosis
   b) *Neurological*
      Coma alcoholicum
      Methanol-induced retrobulbar optic neuritis
   c) *Internal medical*
      Liver function disturbances
      Alcoholic steatosis hepatis
      Acute alcoholic gastritis
      Acute alcoholic (gastro-)enteritis
      Acute alcoholic pancreatitis
      Mallory-Weiss syndrome
      Starvation ketosis
      Alcoholic hyperuricemia
      Methanol intoxication
      Shock due to disulfiram-ethanol reaction
      Hyperlipemia
      Hypercholesterolemia
      Flattened G T T
      Metabolic lactic acidosis

3) *Syndromes and symptoms due to chronic intoxication*
   a) *Psychiatric*
      Chronic alcoholism
      Chronic alcoholism, Skid Row type
      Poriomania
      Alcoholic paranoia
      Pathological marital jealousy
      Alcoholic dementia
      Impotentia coeundi
      Severe marital problems
      Sexual promiscuity
      Prostitution
      Common law relationships
      Attempted suicide
      Suicide
   b *Neurological*
      Korsakoff Wernicke syndrome due to thiamine deficiency
      Korsakoff-Wernicke syndrome due to vit B_{12} deficiency
      Alcoholic polyneuropathy
   c *Internal medical*
      Alcoholic liver cirrhosis
      Hemorrhage of esophageal varices in liver cirrhosis
      Ascites due to liver cirrhosis
      Hepatic coma due to liver cirrhosis
      Suspected hepatoma
      Chronic atrophic gastritis
      Chronic atrophic gastritis with vit B_{12} deficiency

Achylia gastrica
Beriberi
Pellagra
Ferriprive anemia
Megaloblastic anemia
Arrhythmia due to alcoholic cardiomyopathy
Myalgia possibly due to alcoholic myopathy

4) *Syndromes and symptoms conducive to excessive drinking*
Escape drinking
Severe marital problems
Sexual promiscuity
Prostitution
Common-law relationships
Alcoholic depression
Alcoholic aggressive and psychopathiform behavior
Alcoholic dementia
Chronic alcoholism
Chronic alcoholism, Skid Row type
Poriomania
Alcoholic paranoia
Pathological marital jealousy
Impotentia coeundi
Premenstrual tension syndrome
Korsakoff-Wernicke syndrome
Heilbronner's pathological intoxication
Socio-economic stress
All alcohol withdrawal syndromes observed
All psychiatric syndromes observed

Table 7 2

Miscellaneous psychiatric, neurological, internal medical, dermatological and surgical diagnoses

1) *Psychiatric diagnoses*
Reactive depression
Vital depression
Agitated depression
Acute agitation
Arteriosclerotic agitation
Arteriosclerotic dementia
Psychopathiform behavior
Pyromania
Sexual exhibitionism
Sexual delinquency
Latent homophilia
Schizoid psychasthenic personality
Schizoid psychosis
Schizophrenia
Psychogenic psychosis
Degenerative psychosis
Psychosis due to mental retardation
Mental retardation
Hysterical conversion
Hysteriform behavior
Bonnhoeffer's hyperesthetic-emotional syndrome
Carcinophobia
Anxiety neurosis
Tension state

2) *Neurological diagnoses*
(Grand Mal) epilepsy
Twilight state
Radial nerve neuritis
Guillain-Barré syndrome
Cerebral thrombosis
Medullary syndrome due to cerebral thrombosis
Transitory ischemic attack
Hypertensive encephalopathy
Epidural hematoma due to traffic accident
Extrapyramidal syndrome due to flufenazine

3) *Internal medical diagnoses*
Hypertension
Generalized arteriosclerosis
Pulmonary emphysema
Bronchopneumonia with pulmonary edema
Extinct pulmonary tuberculosis
Urinary tract infection
Prostatic hypertrophy
Renal insufficiency
Status post Billroth II gastrectomy
Dumping syndrome
Cardiospasm
Constipation
Diabetes mellitus
Hyperglycemic ketoacidotic coma
Premenstrual tension syndrome
Gout
Exogenous obesity
Acute tonsillitis
Acute rheumatic fever
Erysipelas
Influenza
Bacillary dysentery
Amebic dysentery
Ascaris lumbricoides infestation
Strongyloides stercoralis infestation
Giardia lamblia infestation
Beta-thalassemia minor
Sickle-cell anemia
Sickle-cell trait

4) *Dermatological-venereological diagnoses*
Latent syphilis
Candida dermatitis
Athlete's foot
Varicellae
Psoriasis vulgaris
Toxicodermia due to phenothiazines

5) *Surgical diagnoses*
Chondromatosis cubiti
Atheromatous cyst on forehead
Old pertrochanteric fracture

Patients were recorded at every hospital admission or at every consultation at the alcoholism outpatient clinic. Their ages were

recorded in this study according to the date of last hospitalization, last visit to the outpatient clinic, or according to the date when they were interviewed for this study.

Hospitalization took place at the former *San Pedro Hospital* in Oranjestad. This was a general hospital with no clearly defined wards, with the exception of the Pediatric and Obstetric wards. The hospital had an intensive care unit for cardiac and other emergencies, several well-equipped operating rooms, delivery rooms and two isolation-cells for severely psychotic patients. The hospital was closed in November 1976 and replaced by the newly built 300-bed Dr. Horacio Oduber Hospital.

When hospitalized, alcoholic patients found themselves surrounded by patients with medical, surgical, neurological, psychiatric, gynecological, dermatological, otorhinological, ophthalmological and other diseases. As far as possible, however, attempts were made at keeping alcoholic patients together in the same rooms.

The *outpatient clinic* for alcoholic, psychiatric and neurological patients was housed in the Public Health building at a 100 metres distance from the hospital. From October 1969 through September 1970 patients were seen on an individual basis at this outpatient clinic; consulting hours were on Tuesdays and Thursdays from 10.30 a.m. to 13.00 p.m. From October 1970 through September 1972 special consulting hours were held for alcoholics on Wednesdays from 10.30 a.m. to 13.00 p.m. This was done in the style of a group therapeutic session: all patients were seen together and in the presence of two AA officers; attention was paid to every patient's particular problem(s) so that all those present could profit maximally; simultaneously prescriptions were renewed. Patients who so desired, could in addition get individual consultations. Remarkably few made use of this possibility.

Hospitalized alcoholics were seen during the daily rounds. After October 1970 most hospitalized alcoholics participated in group therapy, which was held twice weekly on Tuesdays and Thursdays from 17.00 p.m. to 19.00 p.m. in one of the hospital's classrooms for the training of nurses, in cooperation with AA officers and other AA members.

7.2. THERAPEUTIC REGIMENS

Medical treatment has become one of the prime factors in recovery

for alcoholics (Block, 1962; Van Epen, 1974; Victor & Adams, 1974). The reasons for this are twofold:

a) Because many alcoholics have a fear of being faced with any psychiatric treatment, a medical approach seems more acceptable;
b) When physical well-being is regained, any type of further therapy is less of a threat, and the cooperation of the patient increases (Block, 1962).

The most important features of treatment are:

1) Thorough diagnostic procedures;
2) Detoxification;
3) Treatment of withdrawal syndromes;
4) Treatment of alcoholismic complications and other ailments;
5) Treatment of alcohol addiction:
   a. Aversion treatment with disulfiram;
   b. Aversion treatment with emetine and/or apomorphine;
   c. Disulfiram maintenance treatment;
   d. Citrated Calcium Carbimide (C.C.C.) maintenance treatment;
   e. Metronidazol maintenance treatment;
   f. Group psychotherapy;
   g. Individual psychotherapy, e.g. psychoanalysis and psychological aversion treatment;
   h. Psychodrama, sociodrama;
   i. Hypnosis;
   j. Disulfiram implantation;
   k. Participation in organizations combating alcoholism, e.g. Alcoholics Anonymous.

(Block, 1962; Esser, 1967; Fox, 1967, 1968; Hoff, 1967; Ketel, 1963; Krafft, 1974; Pawan, 1970; Pinto, 1963; Rankin, 1969; Santamaria, 1969; Van Epen, 1974; Van Erp, 1971; Victor & Adams, 1974). The requisite for successful treatment is total abstinence; this presents the only permanent solution, notwithstanding sporadic statements concerning minorities of alcoholics presumably returning to moderate (social) drinking (Bailey & Stewart, 1967; Canavan, 1971; Davies, 1962; Davies et al., 1969; Fox, 1967, 1968, 1971; Victor & Adams, 1974; Sijlbing, 1977).

Patients hospitalized for alcoholism (or its precursor stages) in the October 1969-September 1972 period were treated according to the following therapeutic regimen:

1) *Diagnostic procedures*
   a) Medical history, including drinking history;
   b) Physical examination;
   c) Laboratory tests (urinalysis; hemoglobin; hematocrit; leukocyte count; platelet count; sedimentation rate; liver function tests; glucose; electrolytes; renal function tests);
   d) Extension of diagnostic assessments (e.g. chest X-ray, ECG) if necessary.

It was kept in mind that the smell of alcohol on the breath does not exclusively point to alcohol intoxication; other types of intoxication, head injuries, diabetic coma, hypoglycemic coma, epileptic states, cerebrovascular accidents, meningo-encephalitis, were ruled out before the diagnosis alcohol intoxication was established.

2) *Detoxification*

This implies the abrupt stopping of alcohol intake, popularly called "sobering up". In the *addicted* alcoholic, detoxification is applied at the risk of provoking the onset of any of the alcohol withdrawal syndromes. It is useless to attempt further treatment such as aversive therapy, or (group) psychotherapy, while the patient is still intoxicated or in withdrawal, since there is no gnostic or ethical judgment present for him to use in order to profit from further therapeutic measures (Block, 1962). Generally, the process of detoxification took from 1 to 10 days (average 5 days).

Coma alcoholicum, a medical emergency, requires two specific measures: a) precention or treatment of respiratory depression; b) prevention or treatment of circulatory collapse (Victor & Adams, 1974).

Heilbronner's pathological intoxication requires adequate sedation, e.g. with phenobarbital 200 mg. subcutaneously, repeated every 30-40 min. if necessary (Victor & Adams, 1974).

Methanol intoxication requires four specific measures: a) Gastric lavage in the first two hours; b) $NaHCO_3$ intravenously to correct metabolic acidosis; c) Ethanol (0.5 ml. per kg. body weight) intravenously every two hours, which may inhibit the metabolism of methanol to its extremely toxic metabolite formaldehyde by the fact that alcohol dehydrogenase, which metabolizes both methanol and ethanol, has a metabolic preference

for ethanol (Koch-Weser, 1974; Merry, 1971); d) Hemodialysis (Koch-Weser, 1974).

3) *Treatment of withdrawal syndromes*

The main purpose in treating withdrawal syndromes is to regain control over the general hyperstimulation of CNS cells caused by withdrawal, by using drugs with sedative effects upon the CNS, in daily decreasing dosages until a new equilibrium is attained (Block, 1962; Ehik, 1968; Gant, 1968; Kamphuisen et al., 1965; Victor & Adams, 1974; Victor & Wolfe, 1973). The basic scheme of action in the treatment of withdrawal is as follows:

a) *Detection of concomitant complications.* E.g. head injuries, pneumonia, tuberculosis, subdural hematoma or meningo-encephalitis. Chest X-rays, skull X-rays, lumbar punctures, and continuous registration of blood pressure, pulse and temperature may be necessary.

b) *Treatment of shock:* fluid replacement, blood transfusions, vasopressor drugs.

c) *Treatment of hyperthermia:* e.g. cooling mattress.

d) *Treatment of concomitant infections,* i.e. antibiotics.

e) *Correction of fluid and electrolyte imbalance.* Severe perspiration may require even 6,000 ml. fluid daily, of which 1,500 ml. should be saline solution.

f) *Treatment of hypoglycemia:* glucose 5 % infusions.

g. *Vitamin B complex* intravenously, later orally.

h) *Sedative drugs:*

h)1. *Chlordiazepoxide* (Librium). The most frequently used sedative among our patients. Dosages varied from 100-400 mg. daily, decreasing to maintenance dosages of 20-40 mg. daily. No side-effects were observed. Oral administation.

h)2. *Diazepam* (Valium). Intramuscular or oral administration. Dosages: 20-40 mg. daily, decreasing to 5-10 mg. daily.

h)3. *Promazine* (Sparine).

h)4. *Oxazepam* (Seresta, Serepax).

h)5. *Thioridazine* (Melleril).

i. *Treatment of alcoholic convulsions:*
   i)1. *Diphenylhydantoin* (Dilantin).
   i)2. *Diazepam* (Valium).
   i)3. *Primidon* (Mysolin).
   i)4. *Carbamazepine* (Tegretol).

j) *Treatment of insomnia:*
   j)1. *Nitrazepam* (Mogadon).
   j)2. *Pentobarbital* (Nembutal).

k) *Treatment of hypomagnesemia:* $MgSO_4$ i.v. proved to be effective in some cases of alcohol withdrawal associated with hypomagnesemia (Kamphuisen et al., 1965; Victor & Wolfe, 1973).

4) *Treatment of alcoholismic complications and other ailments*

a) *Correction of dehydration or overhydration.*
In many cases of prolonged alcohol intake, clinically evident *dehydration* occurs due to: hyperhidrosis (concomitant with cutaneous vasodilatation); water diuresis due to transient suppression of ADH release; and vomiting and/or diarrhea from alcoholic gastritis and gastroenteritis. Correction is possible with glucose 5 %, glucose-saline, saline, and fructose 5 %-10 %. Especially in cases of coma alcoholicum and delirium tremens fructose appeared to be effective (Pawan, 1970; Van Epen, 1974). *Overhydration* is less common, and may be due to congestive heart failure; it may be corrected by fluid and sodium restriction, diuretics and digitalis.

b) *Correction of fluid and electrolyte imbalance.*
Guidelines: clinical picture, volume of diuresis, serum electrolytes.

c) *Correction of vitamin, mineral, protein and other deficiencies.*
Hypoglycemia, hypokalemia, hypomagnesemia, hypalbuminemia due to cirrhosis, and multivitamin deficiencies are frequently observed, and require substitution.

d) *Appropriate dietetic measures.*
   d)1. *Sippy or Meulengracht diet* for patients with alcoholic gastritis or peptic ulcer.
   d)2. *Low fat, high protein, high carbohydrate diet* for patients with liver disease (Block, 1962; Hoff, 1967; Gerrits & Vandenbroucke, 1971).

d)3. *Low salt diet* for patients with overhydration, hypertension, congestive heart failure.

d)4. *Low protein diet* for patients with hepatic coma.

d)5. *Diabetes diet* (low carbohydrate) for diabetics.

e) *Treatment of depressive states.*

Depressive states are often seen during intoxication or withdrawal. These were treated with *dibenzazepine*-derivatives, such as *imipramine* (Tofranil) or *trimipramine* (Surmontil) or *amitryptiline* (Elavil).

f) *Treatment of psychoses.*

These were treated with *phenothiazines,* such as *thioridazine* (Melleril), *chlorpromazine* (Largactil) or with *haloperidol* (Serenase).

5) *Treatment of alcohol addiction.*

5)a) *Aversion treatment with disulfiram.*

Disulfiram (Refusal, Antabus, Esperal) is a drug designed to keep the alcoholic from drinking. Ingestion of ethanol while under this therapy results in an episode of severe illness, which is so disquieting that knowledge of such a reaction acts as an excellent deterrent. Disulfiram *alone* has little effect on the body: fatigue, headache, slight decrease of sexual potency, occasionally a skin allergy (Block, 1962). Mention has been made of lactic acidosis (Williams, 1974).

5)a)1. *The Disulfiram-Ethanol-Reaction (D.E.R.)*

After ingestion of slight amounts of alcohol, the Disulfiram-Ethanol-Reaction (D.E.R.) develops. The D.E.R. can be subdivided arbitrarily in the following types:

(1) *Acute effects.*

(a) Mild reaction: flushing, hyperhidrosis, conjunctival hyperemia, heat sensations, slight rise of peripheral temperature, nausea, tremor, slight edema of face and neck, rise in blood pressure, generalized pruritus, urticaria-like exanthema.

(b) Moderate reaction: vomitus, tachycardia, hyperventilation, pounding headache, dizziness, restlessness, paresthesia, rhinorrhea, feelings of exhaustion, "poisonous" taste.

(c) Severe reaction: somnolence, confusion, psychosis, respiratory depression, cyanosis, hypotension, shock, death.

(2) *Late effects.*

Slight decrease of libido and potency, polyneuropathy, loss of appetite, fatigue, headache, skin allergy (delayed type), weight loss, liver damage, psychasthenia, psychosis (i.e. depression, paranoia, manic psychosis, Korsakoff psychosis), death.

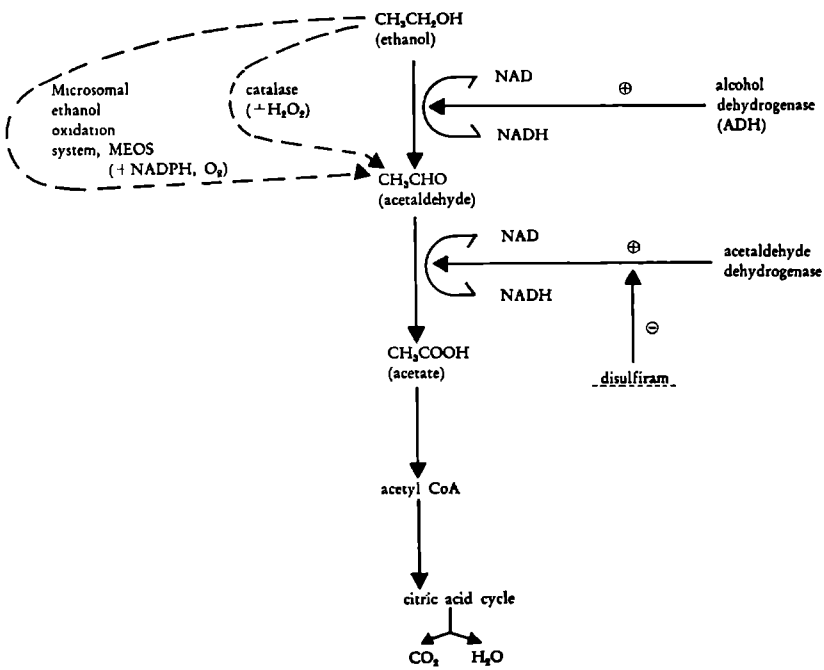
5)a)2. *Nature of the D.E.R.*

The true nature of the D.E.R. is not fully understood as yet. The two most widely accepted explanations are as follows:

(a) *Acetaldehyde dehydrogenase inhibition.*

Inhibition of acetaldehyde dehydrogenase induces increased serum acetaldehyde levels (Merry, 1971; Truitt & Duritz, 1967).

Increased serum acetaldehyde levels induce: metabolic



stimulation and release of catecholamines in adrenal medulla and brain; interference with pyruvate maintenance of mitochondrial oxidative phosphorylation (Isselbacher, 1977; Truitt & Duritz, 1967; Walsh, 1973); lactic acidosis (Isselbacher, 1977; William, 1974). This happens along the pathways in oxidation of ethanol by the liver, as shown on page 208.

(b) *Direct toxic effects of disulfiram or its metabolites.* Disulfiram inhibits tissue respiration and consequently induces lactic acidosis (Perman, 1962; Williams, 1974).

5)a)3. *Technique of aversion treatment with disulfiram.*

Patients received disulfiram (Refusal or Antabus) 1500 mg. for 4 days, followed by 500 mg. on the 5th and 6th day. On the 7th day 250-500 mg. of disulfiram was given in the morning, followed by testdrinks in the afternoon, i.e. 500 ml. of beer or 200 ml. of wine of 50-100 ml. of distilled spirits; this procedure was repeated 5-8 times on alternating days. The testdrink is generally followed by the D.E.R. within 10-90 minutes, which fades away in about half a day. The most dangerous effects of the D.E.R., i.e. shock and respiratory depression, were treated with dexamethason i.v., adrenaline i.v., levarterenol i.v., polygeline and saline infusions, and oxygen and prethcamide respectively.

Among 24 patients who received this type of aversion treatment, 2 showed no reaction, 3 a mild reaction, 15 a moderate reaction, and 4 a severe reaction (1 went in deep shock). These unfavorable experiences were the reason to stop this type of aversion treatment after September 1970. Moreover, it is no longer considered necessary to demonstrate the D.E.R. effects to the patient, provided that he is warned about the severe reactions that may result if he drinks while disulfiram is in his system (Victor & Adams, 1974; Victor & Wolfe, 1973).

5)a)4. *Contraindications for disulfiram treatment.*

(1) Liver cirrhosis

(2) Other major liver diseases (hepatoma, metastatic disease, hepatitis etc.)

(3) Renal diseases (renal insufficiency, nephritis, etc.)
(4) Cardiac diseases (congestive heart failure, coronary disease, vitia cordis, myocardiopathies, etc.)
(5) Vascular diseases (arteriosclerosis, morbus Bürger, etc.)
(6) Hypertension
(7) Diabetes mellitus
(8) Respiratory diseases (chronic obstructive lung disease, restrictive lung disease, etc.)
(9) Epilepsy
(10) Malnutrition
(11) Drug addiction
(12) Dementia
(13) Psychosis
(14) Pregnancy

5)b) *Aversion treatment with apomorphine and/or emetine.*
This type of aversion treatment implies *conditioned response therapy* which is based on the theory that the individual patient can be trained to a particular psychological reaction under certain defined situations.
Apomorphine HC1 5-10 mg. s.c. was administered to the patient, or emetine 10-20 mg. s.c. or i.m., followed by testdrinks. After apomorphine testdrinks are generally followed within 10-30 minutes by the direct effects of apomorphine: nausea, sialorrhea and vomiting (by stimulation of the emetic center in the brainstem). In this series of patients *none* of the following side-effects of apomorphine were seen: tremor, restlessness, hyperventilation, convulsions, hypotension or coma. After emetine testdrinks are generally followed within 10-30 minutes by the direct effects of emetine: nausea and vomiting. *None* of the side-effects of emetine (cardiac arrhythmia, hypotension) were seen. Patients who did not respond satisfactorily (i.e. vomiting) to apomorphine were switched over to emetine, and vice versa. There are no contraindications for apomorphine, and for emetine only one: cardiac disease.
Before October 1970 some patients received aversion treatment with disulfiram plus apomorphine or emetine

plus testdrinks, especially those in whom vomiting did not occur on disulfiram alone. Because of the untoward experiences as described previously, this type of treatment was discontinued after October 1970.

Aversion treatment with apomorphine and/or emetine has been applied from October 1969 through September 1972; it has substituted entirely aversion treatment with disulfiram after October 1970.

5)c) *Disulfiram maintenance treatment.*

To all patients without contraindications and who were willing to accept this drug, disulfiram (Refusal, Antabus) was prescribed orally 250-500 mg. daily, after warning them about the effects of disulfiram during alcohol ingestion (Block, 1962; Esser, 1967; Fox, 1967, 1968; Victor & Adams, 1974; Victor & Wolfe, 1973).

5)d) *Citrated calcium carbimide (C.C.C.) maintenance treatment.*

This drug has not been applied because of its unavailability in Aruba. It acts in the same way as disulfiram. Advantages are that some of the discomforts found with disulfiram (e.g. sexual impotence, allergic dermatitis, somnolence) do not occur. One disadvantage is, that the C.C.C. effect maximally lasts for 18 hours, while the disulfiram effect holds for at least 24 hours (Esser, 1967).

5)e) *Metronidazol (Flagyl) maintenance treatment.*

Commonly used in the treatment of Trichomonas vaginalis, this drug appeared to provoke an aversion against alcohol in some alcoholics: it would add a metallic flavor to beverage alcohol (Van Epen, 1974). However, it appears to have poor therapeutic results (Fox, 1967, 1968; Ditman, 1968; Van Epen, 1974). It was administered to only one patient, unsuccessfully.

5)f) *Group therapy.*

Group therapy has been applied from October 1970 through September 1972 (Fox, 1967, 1968; Leach, 1973; Pinto, 1963). Most hospitalized patients agreed to participate. Patients were brought to group therapeutic sessions after detoxification, treatment of withdrawal syndromes

and of complications, and after aversive treatment was started. Group therapy was performed in dialogue with AA officers and other AA members.

The main characteristics of this type of group therapy are as follows:

(1) Non-psychoanalytical;

(2) Explanatory; i.e. extensive explanation was given to patients regarding the diagnostic features of alcoholism, problem drinking, alcohol intoxication, tolerance, physical dependence, withdrawal syndromes, loss-of-control, craving, psychological dependence, toxic effects of alcohol, and various etiological theories such as social and psychological circumstances conducive to alcoholism;

(3) Family-centered; i.e. wives, husbands, children and parents of hospitalized alcoholics were also urged to participate in discussions of presented themes, and patients and their relatives present were stimulated to express and discuss their conjoint problems and to propose their own views concerning solutions thereof;

(4) Supportive; i.e. moral support was given to patients in need of such, while problem-solving support was given whenever possible;

(5) Indoctrination by Alcoholics Anonymous (AA).

5)g) *Individual psychotherapy.*

Individual psychotherapy (Fox, 1967, 1968; Ditman, 1968) such as psychoanalysis and Rogerian psychotherapy, was considered the field of the psychiatrist (or psychotherapist). Since these were not present in Aruba from July 1969 to August 1972, these types of treatment were not applicable. Psychoanalysis has had poor treatment results in other studies (Fox, 1967, 1968; Ditman, 1968).

5)h) *Psychodrama, sociodrama.*

These types of treatment were considered the field of the psychiatrist (or psychotherapist), and hence not applied.

5)i) *Hypnosis.*

This type of treatment was considered the field of the psychiatrist (or psychotherapist), and hence not applied.

Hypnosis has had poor treatment results in other studies (Fox, 1967, 1968; Ditman, 1968).

5)j) *Disulfiram implantation.*

This type of treatment was given to 20 patients, in whom repeated aversion treatment and group therapy did *not* give satisfactory results. It implies the subcutaneous or subfascial (under the fascia of the m. rectus abdominis) implantation of 10 Esperal (disulfiram 100 mg.) pellets. In our patients Esperal pellets were implanted subcutaneously in the left upper quadrant of the abdominal wall about 10 cm. below the left costal arch. In this type of treatment the contraindications for disulfiram therapy have to be considered stringently. Esperal pellets were obtained from Laboratoires Thersa, France. Disulfiram implantation was applied for the first time, not only in Aruba but in the Netherlands Antilles, on June 9, 1971. Implantations were performed - after the indication had been established by the author - by one of the surgeons A. J. C. Hazenberg and F. M. A. Saladín. A sterile metal tube 15 cm. in length is brought into the subcutis under local anesthesia after a short incision of 1-2 cm. One by one the Esperal pellets are deposited with a mutual distance of 1-2 cm. to prevent conglomeration. After the implantation procedure the incision is closed. All implanted patients also received aversion treatment and group therapy before implantation and oral disulfiram maintenance after discharge from hospital, as well as help from AA. The implantation was presented to them as functioning as a "safety belt".

Disulfiram implantation has been introduced in France, and repeated afterwards in several other countries (Hrynkiewicz et al., 1966; Kellam, 1969; Kellam & Wesolkowski, 1968; Kraft, 1974; Malcolm, 1972; Marie, 1955; Paillot & Jacques, 1968; Prigent, 1960; Van Erp, 1971). The Esperal pellets were expelled after suppuration in one patient, due to the fact that he tried to remove them by hand. No other somatic reactions were seen after implantation. A possibly disulfiram-induced psychosis was

observed in one of the 20 implanted patients (pat. no. 30150 in table 7.9.), of the paranoid and depressive type (Kraft, 1974).

Indications for Esperal implantations mentioned in the literature: "revolving door" alcoholism (Kellam & Wesolkowski, 1968; Kraft, 1974; Malcolm, 1972); alcoholics who have failed to maintain taking disulfiram orally (Keller & Wesolkowski, 1968; Kraft, 1974); intelligent neurotic alcoholics in whom with the aid of implantation there could be attempted to restore defense mechanisms (Kraft, 1974).

Contraindications mentioned in the literature: those applicable for oral disulfiram; previous intolerance of oral disulfiram (Malcolm, 1972); alcoholics living alone (Malcolm, 1972); neurotic alcoholics (Kraft, 1974); psychopathic alcoholics (Kraft, 1974); epsilon alcoholism (Kraft, 1974).

5)k) *Participation in organizations combating alcoholism, e.g. Alcoholics Anonymous (AA).*

(Bailey & Leach, 1965; Block, 1962; Canavan, 1971; Esser, 1967; Fox, 1967, 1968; Leach, 1973; Van Epen, 1974; Victor & Adams, 1974; Victor & Wolfe, 1973). In Aruba the only organization combating alcoholism open to alcoholics, is *AA*. Members of AA were stimulated to participate in group therapy and to visit hospitalized alcoholics in order to persuade these patients to accept the AA program and to become AA members. This contact between recovered AA members and hospitalized alcoholics is deemed beneficial as a counter-pressure against the demoralization due to negative attitudes of non-alcoholic patients, nurses, visitors and hospital medical personnel towards alcoholics (Ehik, 1968; Esser, 1965; Pinto, 1963). AA has proved to be the single most effective force in the rehabilitation of alcoholic patients (Bailey & Leach, 1965; Leach, 1973; Norris, 1976; Victor & Adams, 1974; Victor & Wolfe, 1973). The philosophy of AA is embodied in their so-called "*twelve steps*" and "*twelve traditions*", a series of propositions about alcohol

and alcoholism which guide the patient to recovery. The AA philosophy stresses in particular the practice of making restitution, the necessity to help other alcoholics, trust in God, the group confessional, and the belief that the alcoholic is powerless over alcohol. The AA philosophy also embodies the "24 hour plan", in which the alcoholic strives for just 24 hours of abstinence, as a means of facilitating the maintenance of sobriety; this purpose is renewed day after day (Victor & Adams, 1974). It has been stated, that about 50 % of the members who express more than a passing interest in the AA program have *no* relapses, and that a significant additional number relapse but eventually recover (Victor & Adams, 1974).

The methods used by AA are *not* suited to every alcoholic; some prefer the more personalized approach offered by special clinics and centers for the treatment of alcoholism. The physician should therefore be aware of all the community resources available for the management of alcoholism, and should be prepared to take advantage of them in appropriate cases (Victor & Adams, 1974).

Of course there are more methods of treatment for alcoholism. (Block, 1962; Ditman, 1968; Esser, 1967; Fox, 1967, 1968; Van Epen, 1974; Victor & Adams, 1974).

Lysergic acid (LSD) has been employed, but with poor therapeutic results (Block, 1962; Ditman, 1968; Fox, 1967, 1968). Not applied in Aruba.

ACTH and corticosteroid hormones, formerly advocated in the management of delirium tremens (Block, 1962), appear to have *no* place in the treatment of alcohol withdrawal syndromes (Victor & Adams, 1974). Not applied in Aruba.

Digitalis is advocated in the treatment of delirium tremens (Van Epen, 1974). Not applied in Aruba, since delirium tremens generally was not accompanied by cardiac failure.

Vitamin B_{12} was given to a few patients with hyperchromic macrocytic anemia in chronic gastritis.

Pancreatine (Combizym, Trizymal) was given to a few patients with alcoholic pancreatitis.

Ferrofumarate orally or *ferrosorbitol-complex* i.m. was given to a few patients with iron deficient anemia.

Antacids were given to some patients with alcoholic gastritis or peptic ulcer.

Anti-emetics (difenhydramine only) were given to patients with severe vomiting.

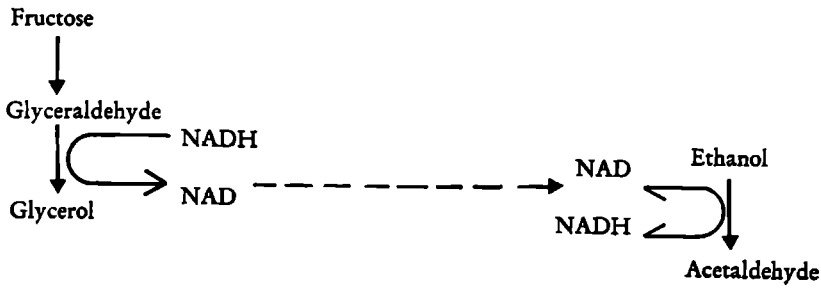
Insulin was given to a few patients with concomitant diabetes.

Dextrose-5 % was generally applied as basic infusion.

Saline was generally applied in cases of electrolyte loss, dehydration and D.E.R. shock.

Albumin was only applied in a few cases of hypalbuminemia in liver cirrhosis.

Fructose-5 % was applied in some cases of alcoholic coma and delirium tremens. Fructose appears to produce a material increase in the rate of alcohol metabolism (Merry, 1971) as shown below:



The effect of fructose would be to regenerate NAD for the metabolism of ethanol.

7.3. FACTORS AFFECTING THERAPEUTIC RESULTS AND PROGNOSIS

There is a need to obtain more knowledge about factors which might affect therapeutic results and hence prognosis, whether beneficiously or unfavorably, in order to ameliorate therapeutic results in the future (Blaney et al., 1975; Mindlin, 1959; Rathod et al., 1966).

The following factors were evaluated as regards their influence on therapeutic results and prognosis:

1) *Factors deduced from patient records;*
2) *Predisposing and perpetuating factors* (cf. Chapters 5 & 6).

7 3.1. Factors deduced from patient records

These are the following:

1) *Therapeutic regimens*
   1)1. Among all pathological drinkers studied.
   1)2. Among alcoholics and gamma-prealcoholics.
   1)3. Among chronic alcoholics.
   1)4. Among problem drinkers.
2) *Diagnostic categories*
3) *AA membership*
4) *Nationality*
5) *Papiamento speaking*
6) *Sex*
7) *Age*
8) *Number of hospitalizations*
9) *Policlinical or clinical treatment*
10) *Complications*
   10) 1. Number of complications.
   10) 2. Alcoholism-with-complications.
   10) 3. (Degree of) liver damage.
   10) 4. Gastritis.
   10) 5. Pancreatitis.
   10) 6. Convulsions.
   10) 7. Polyneuropathy.
   10) 8. Psychosis.
   10) 9. Hallucinosis.
   10)10. Delirium tremens.
   10)11. Hypomagnesemia.
   10)12. Flattened G.T.T.
   10)13. Korsakoff-Wernicke.
   10)14. Methanol intoxication.
   10)15. Hypertension.

All these factors were cross-tabled with the factor "*objective evaluation therapeutic results*", consisting of one variable with a five-point score; cf. *table 7 3*

Table 7.3.

Objective evaluation therapeutic results.

Therapeutic Result	Score
Dead (D)	1
Not sober (NS)	2
Lost to follow-up (L)	3
Semi-sober (SS)	4
Sober (S)	5

The evaluation of therapeutic results was not done by the author, but by two AA-officers, one nurse and one social worker, all employed by the government on behalf of the outpatient clinic for alcoholics.

The following factors appeared to correlate significantly with the factor "objective evaluation of therapeutic results":

1) Therapeutic regimens among all pathological drinkers.
2) Therapeutic regimens among alcoholics and gamma-prealcoholics.
3) Diagnostic categories.
4) AA-membership.
5) (Degree of) liver damage.

These significant correlations are shown in *table 7.4.*

The following reasons can be given for the choice of therapeutic regimens:

a) Treatment was considered contraindicated in the presence of serious somatic or mental disease of any type, or if sufficient contraindications for disulfiram therapy were present;
b) The "treatment refused" category is self-evident;
c) Disulfiram maintenance treatment was the therapy of choice for patients only treated at the outpatient clinic or briefly hospitalized who had no disulfiram contraindications and who refused group therapy;
d) Aversion treatment was the therapy of choice for hospitalized patients before October 1970, and after that date for a few who refused group therapy;
e) Aversion treatment plus group therapy was the therapy of choice

for hospitalized patients after October 1970, who had no disulfiram contraindications;

f) Group therapy was the therapy of choice for hospitalized patients with disulfiram contraindications after October 1970;

g) Disulfiram implantation was applied for "revolving door" patients who had no (or very few) disulfiram contraindications and who had failed repetitively on oral disulfiram.

Table 7.4.

Factors affecting therapeutic results and prognosis.
I) Factors deduced from patient records. Therapeutic results in percent per factor.

Factor	N	Therapeutic Results						χ^2	df	p
		D*	NS*	L*	SS*	S*	Mean therapeutic score			
1) *Therapeutic regimens among all pathological drinkers*								35.02	6	<0.005
Treatment contraindicated	27	37	11	26	7	19	2.6			
Treatment refused	36	3	8	44	25	20	3.5			
Disulfiram maintenance	32	0	9	25	28	38	4.0			
Aversion treatment	19	4	32	16	32	16	3.3			
Group therapy	41	2	2	17	25	54	4.3			
Aversion treatment & Group therapy	73	3	15	8	37	37	3.9			
Esperal implantation, preceded by Aversion treatment & Group therapy	20	15	0	0	45	40	4.0			
Total	248	7	11	19	29	34	3.7			
2) *Therapeutic regimens among alcoholics & gamma-prealcoholics*								20.02	6	<0.005
Treatment contraindicated	10	40	0	20	20	20	2.8			
Treatment refused	21	0	13	29	29	29	3.7			
Disulfiram maintenance	26	0	8	27	31	34	3.9			
Aversion treatment	12	0	25	17	33	25	3.6			
Group therapy	28	0	4	4	25	67	4.6			
Aversion treatment & Group therapy	59	2	15	7	37	39	4.0			
Esperal implantation, preceded by Aversion treatment & Group therapy	15	6	0	0	47	47	4.3			
Total	171	4	11	13	32	40	3.9			

3) *Diagnostic categories*								29.95	3	<0.005
Problem Drinking	47	0	9	49	21	21	3.5			
Gamma-prealcoholism	9	0	33	22	33	12	3.2			
Alcoholism	162	4	9	12	33	42	4.0			
Chronic alcoholism	30	40	17	6	20	17	2.6			
4) *AA-membership*								54.96	1	<0.005
Membership	135	4	10	2	37	47	4.1			
No membership	113	12	12	38	19	19	3.2			
5) *Degree of liver damage*								11.10	2	<0.005
No liver damage	115	5	10	29	25	31	3.7			
Slight liver damage (liver function disturbances with or without hepatomegaly; steatosis)	122	5	12	11	34	38	3.9			
Severe liver damage (cirrhosis; suspected hepatoma)	9	67	0	0	22	11	2.1			

χ^2 is computed for (S + SS) versus (D + NS + L). * D = dead; NS = not sober; L = lost to follow-up; SS = semi-sober; S = sober.

As shown in *table 7.4.*, the following conclusions may be drawn:

1) *Therapeutic regimens among all pathological drinkers.*
The results indicate, that the therapeutic regimens introduced after October 1970 have had definitely more success than those before October 1970. Those newer therapeutic regimens, introduced since October 1970 are:
   — Group therapy;
   — Aversion treatment *plus* group therapy;
   — Esperal implantation (after aversion treatment *plus* group therapy).
2) *Therapeutic regimens among alcoholics and gamma-prealcoholics.*
The same conclusion may be drawn as sub 1).
3) *Diagnostic categories.*
The results indicate that therapeutic results under the therapeutic regimens applied, are *best* for *alcoholics, worst* for *chronic alcoholics,* with *problem drinkers* and *gamma-prealcoholics* somewhere in between.
The differences in therapeutic results between the diagnostic categories are provoked however by two different mechanisms:
   a. the *higher mortality rate* among chronic alcoholics;
   b. the *lower degree of motivation* among problem drinkers and gamma-prealcoholics, judged by their higher rates of "lost to

follow-up" and "not sober"; this is in agreement with the view that *these* pathological drinkers have not yet experienced the so-called "hitting the bottom" phenomenon: reaching the point of maximal defeat, at which the need for external help is best acknowledged (Jellinek, 1962; Glatt, 1975) *or* with the possibility that our actual therapeutic regimens may be less able to motivate problem drinkers to sobriety.

4) *AA-membership.*
The results indicate that when AA-membership is present, therapeutic results are better, than in the absence of AA-membership. The differences in therapeutic results between AA-members and non-AA-members are mainly due to the *lower degree of motivation* of the latter judged by their higher rates of "lost to follow-up", though they also appear to have a *slightly higher mortality rate* than AA-members.
The favorable effects of Alcoholics Anonymous for the prognosis of alcoholism are well known (Bailey & Leach, 1965; Leach, 1973; Norris, 1976; Victor & Adams, 1974; Victor & Wolfe, 1973).

5) *Degree of liver damage.*
The results indicate, that therapeutic results are worst for those cases with *severe* liver damage, and that "slight" liver damage does *not* apparently affect treatment outcome, at least not over this *short* three years'period. The differences in therapeutic results between "severe" liver damage and "slight" or "no" liver damage are apparently due to the *higher mortality rate* of severe liver damage.

7.3.2. Predisposing and perpetuating factors (cf. Chapters 5 & 6)
These are the following:

1) *Predisposing factors.*
   1. Paternal inebriety in youth
   2. Interparental conflicts in youth
   3. Inebriety in extended family
   4. Autochthony
   5. Rural domicile
   6. Low educational level
   7. (Male) sex
   8. Economic availability of alcohol

2) *Perpetuating factors.*
   1. Marital conflicts
   2. Low social status of respondent
   3. Poor housing conditions
   4. (Proneness to) social pressure to drink
   5. Sociocultural deprivation Aruban males
   6. Anxiety
   7. Suicidality
   8. Boredom
   9. Introversion
  10. Dissatisfaction
  11. Oral fixation or regression
  12. (Low) religious involvement
  13. Negative relatives' attitudes towards drinker's drinking
  14. Low vs. high AA-involvement

The same factors were studied as in the Risk Factor Analysis for *all respondents.* (cf. Chapter 6). To these were added: "(Low) religious involvement", because religious involvement is highly promoted by AA (Leach, 1973), and was mentioned as a prognostic factor (Rathod et al., 1966); "negative relatives' attitudes towards drinker's drinking", because - though it primarily may be the consequence of drinker's drinking - it may perpetuate excessive drinking; "low vs. high AA-involvement" for reasons already mentioned.

All these factors were cross-tabled with the factor "*objective and subjective evaluation of therapeutic results*", consisting of two parts, i.e.:

a) objective evaluation of therapeutic results (cf. *table 7.3.);*
b) subjective evaluation of therapeutic results (cf. *table 7.5).*

This factor is shown in *table 7.5.*

Table 7.5.
Objective and subjective evaluation of therapeutic results.

Variable	Score
a) *Objective evaluation*	
Dead (D)	1
Not sober (NS)	2
Lost to follow-up (L)	3
Semi-sober (SS)	4
Sober (S)	5
Not applicable	—

b) *Subjective evaluation*

b)1 *Frequency of intoxication after treatment (Variable no 173)*

Never	7
Once yearly or less	6
Only on festive days	4
Once every 2 months	5
At most once a week	3
Every weekend	2
2—3 times a week	2
Every day	1
Not applicable	—

b)2 *Amount of drinking after treatment (Variable no 166)*

Not taken a single drink	7
Made a "slip" once	5
"Slipped" several times	4
Drinking as much or more than before	2
Not applicable	—

Total evaluation

(1) In cases of differences in scores between a) and b), the *lower* of the two scores is accepted

(2) *Score 5* for a) = *Scores 6 or 7* for *var 173*,
or *Score 7* for *var 166*
This is defined as good *therapeutic result*

(3) *Score 4* for a) = *Scores 4 or 5* for *var 173*,
or *scores 4 or 5* for *var 166*
This is defined as moderate *therapeutic result*

(4) Scores 1, 2, 3 for a) = *scores 1, 2 3* for *var 173*,
or *score 2* for *var 166*
This is defined as poor *therapeutic result*

(5) If a) or b) is missing, *total evaluation* is defined on *one* of both

The purpose of the combination of "objective" and "subjective" evaluation of therapeutic results as shown in *table 7.5.* is to arrive at a broader and more modest definition of good therapeutic results.

The factor *"low vs. high AA-involvement"* was evaluated as shown in *table 7 6.*

Table 7 6
AA-involvement

Variable	Score
AA-membership (Variable No 151)	
Yes	1
No	0
Not applicable	0
Frequency of speaking at AA meetings (Variable No 152)	
Always	3
Regularly	2
Occasionally	1
Never	0
Not applicable	0

Frequency of visiting or helping alcoholics in hospital or at home (Variable No. 153)

Regularly	2
Sometimes	1
Never	0
Not applicable	0

Frequency of attending AA meetings (Variable No. 154)

More than 3 times a week	6
1-2 times a week	5
1-2 times a month	4
Monthly	3
1-6 times a year	2
Seldom	1
Never	0
Not applicable	0

Evaluation:
(1) Scores are *added*. Range for total score: 0-12.
(2) *Above median scores* are defined as "*high* AA-involvement".
(3) *Below median scores* are defined as "*low* AA-involvement".

Only 82 respondents were evaluable as regards the factor "objective and subjective evaluation of therapeutic results", as defined in *table 7.5*.

The following factors appeared to correlate significantly with the factor "objective and subjective evaluation of therapeutic results":
1) Negative relatives' attitudes towards drinker's drinking;
2) High vs. low AA-involvement.

These two significant correlations are shown in *table 7.7*.

Table 7.7.
Factors significantly correlated with therapeutic results and prognosis. II) Predisposing and perpetuating factors. Therapeutic results in percent per factor.

Factor	N	Therapeutic Results				χ^2	df	p
		Poor (N = 20)	Moderate (N = 30)	Good (N = 32)	Mean therapeutic score			
1) *Negative relatives' attitudes towards drinker's drinking*						5.72	2	<0.06
Yes	65	22	43	35	3.9			
No	17	35	12	53	3.8			
2) *High versus low AA-involvement*						6.66	2	<0.05
High	41	37	32	32	3.6			
Low	41	12	42	46	4.2			

As may be seen in *table 7.7.*, however, the results are *contrary* to expectation, since it is clear that in this study *poor therapeutic results* are correlated with 1) *absence* of negative relatives' attitudes towards drinker's drinking, and 2) *high* AA-involvement.

The results do not confirm those of other studies (Blaney et al., 1975; Mindlin, 1959; Rathod, 1966), in which an extensive number of factors was found as predictors of therapeutic outcome in alcoholism. Especially the contrasting findings for "high vs. low AA-involvement" are remarkable, since in *table 7.4. AA-membership* was found positively correlated to good therapeutic results (as expected).

7.3.3. Discussion of results.

Considering both *table 7.4.* and *table 7.7.*, it is evident that the only factors truly affecting therapeutic outcome at least over a three years' period are those mentioned in *table 7.4*, i.e.:

1) Therapeutic regimens (versus non-treatment);
2) Diagnostic categories;
3) AA-membership;
4) (Degree of) liver damage.

Since only "diagnostic categories" and "liver damage" become manifest *before* treatment, it is not possible on the basis of our present knowledge to predict therapeutic outcome (i.e. *prognosis)* on the basis of *pre-treatment variables*, at least not in such a short period as three years.

Both *pre-* and *intra*-treatment variables were shown to have *weak* associations with treatment outcome (Blaney et al., 1975). There has been a notable level of contradictory findings (of the kind found in this series of patients) in other studies (Blaney et al., 1975).

That *treatment per se* does have an impact on therapeutic results, is shown in *table 7.8.*

That *(degree of) liver damage* does have an impact on therapeutic results, especially through its higher mortality, is shown in *table 7.9.* (causes of death in eighteen alcoholics).

Table 7 8

Therapeutic results in percent, mean duration of periods before and after accepting treatment, and mean numbers of hospital admissions before and after accepting treatment, among eleven "pathological drinkers"

	Mean number of hospital admissions (& range)	Mean duration of pre- or post-treatment periods (& range)	Therapeutic Results				
			D	NS	L	SS	S
Before accepting treatment	1 6 (1-3)	13 months (1 29)	—	100	—	—	—
After accepting treatment	2 0 (1-5)	16 months (5-33)	27	18	9	37	9

Table 7 9

Causes of death in alcoholics

Patient No	Sex	Age	Deceased in hospital (H) or outside hospital (O)	Obduction	Cause(s) of death	Other diagnoses
30104	M	45	O	—	Traffic accident with rupture of arteria iliaca communis dextra	1) Alcoholism 2) Alcoholic convulsions 3) Transient hypertension 4) Liver function disturbances 5) Alcoholic hypomagnesemia 6) Flattened G T T.
30131	M	52	O	—	Unknown	1) Chronic alcoholism 2) Alcoholic convulsions 3) Left radial nerve neuritis 4) Hypertension 5) Flattened G T T 6) Liver function disturbances
30135	M	40	O	—	Traffic accident, unclassified	1) Alcoholism 2) Flattened G T T.
30139	M	48	H	—	Decompensated liver cirrhosis with ascites, edema, icterus, esophageal varices and hepatic coma	1) Chronic alcoholism 2) Alcoholic convulsions

30147	M	50	O	—	Death from drowning in shallow water, suspected suicide because of depression	1) Alcoholism 2) Depression 3) Liver function disturbances
30148	M	35	O	—	Death from drowning in shallow sea water, possibly during convulsion or possibly suicide	1) Chronic alcoholism 2) Alcoholic convulsions 3) Hypertension 4) Liver function disturbances
30150	M	46	O	—	Unknown	1) Chronic alcoholism 2) Grand Mal epilepsy 3) Extinct pulmonary tuberculosis 4) Mild ferriprive anemia e c i 5) Flattened G T T 6) Possibly disulfiram induced psychosis after Esperal implantation
30155	M	36	H	+	Bronchopneumonia with massive pulmonary edema (during delirium tremens)	1) Alcoholism 2) Delirium tremens
30157	F	58	H	+	1) Hemorrhage in esophageal varices 2) Liver cirrhosis with ascites, edema and icterus, and hepatic coma	1) Chronic alcoholism 2) Alcoholic pancreatitis 3) Alcoholic dementia 4) Ferriprive anemia 5) Flattened G T T 6) Alcoholic hypomagnesia
30167	M	69	O	—	Unknown	1) Chronic alcoholism 2) Alcohol withdrawal syndrome 3) Hypertension 4) Arteriosclerotic dementia 5) Liver function disturbances
30174	M	57	H	—	Brainstem syndrome with profound coma and hyperthermia due to cerebral thrombosis with rightsided hemiparesis	1) Chronic alcoholism
30184	M	65	H	—	1) Renal insufficiency 2) Decompensated liver cirrhosis with hepatorenal syndrome	1) Chronic alcoholism

30186	M	33	O	—	Death from drowning in shallow water (rain-tank in his own yard), suspected suicide because of depression	1) Alcoholism 2) Depression
30194	M	52	H	—	1) Decompensated liver cirrhosis with ascites; 2) Suspected hepatoma	1) Chronic alcoholism 2) Alcoholic polyneuritis 3) Pulmonary emphysema
30196	M	44	H	+	1) Traffic accident with epidural hematoma; 2) Decompensated liver cirrhosis	1) Chronic alcoholism 2) Alcoholic convulsions 3) Alcoholic gastritis 4) Hypertension
30209	M	69	O	—	Unknown	1) Chronic alcoholism 2) Arteriosclerotic dementia 3) Flattened G T T 4) ß-Thalassemia minor
30221	M	57	H	—	Methanol intoxication	1) Chronic alcoholism 2) Delirium tremens 3) Hypertension 4) Urinary tract infection 5) Alcoholic psychosis 6) Liver function disturbances 7) Flattened G T.T. 8) Thiamine deficiency 9) Alcoholic gastritis
30239	F	55	H	—	Decompensated liver cirrhosis with hepatic coma	1) Alcoholism

As shown in *table 7.9.* liver cirrhosis was the most prevalent cause of death among these eighteen alcoholics, except "unknown" causes.

In order of prevalence these causes of death can be specified as follows:

— unknown causes of death (4 cases);
— decompensated liver cirrhosis (3);
— decompensated liver cirrhosis *plus* renal insufficiency (1);
— decompensated liver cirrhosis, suspected hepatoma (1);
— traffic accident with subsequent epidural hemorrhage, *plus* decompensated liver cirrhosis (1);
— traffic accidents (2);
— drowning, suspected suicide (2);
— drowning due to convulsion or possibly suicide (1);

— bronchopneumonia during delirium tremens (1);
— brainstem syndrome due to cerebral thrombosis (1);
— methanol intoxication (1).

It should be mentioned that the four cases with "unknown causes of death" all died weeks or months *after* discharge from hospital - as well as the two traffic accidents and three cases of drowning.

That among the *diagnostic categories chronic alcoholism* has the worst prognosis particularly through its very *high mortality rate* (*40 %* in three years) is shown in *table 7.4.* This accentuates the importance of spending the bulk of energy and finances in preventive and therapeutic measures for problem drinkers, (gamma-)prealcoholics and non-chronic alcoholics, in order to reduce the incidence of the end stage of the disease, i.e. chronic alcoholism.

7.4. THERAPEUTIC RESULTS ACCORDING TO THERAPEUTIC REGIMENS

7.4.1. Therapeutic "gradient"

As shown in *table 7.4.* therapeutic results among all pathological drinkers appear to improve on the "gradient":
treatment contraindicated → treatment refused → aversion treatment → disulfiram maintenance treatment → group therapy plus aversion treatment → group therapy → Esperal implantation.

This same "gradient" is seen among alcoholics & gamma-prealcoholics, as shown in *table 7.4.* To understand this "gradient" the percentages of *S* and *SS* have to be *added.*

The introduction of *group therapy* and *Esperal implantation* after October 1970 probably was a very valuable contribution to the arsenal of therapeutic regimens for alcoholics in Aruba. *Group therapy,* however, seems to be the more important one of these regimens, since (a) all cases of Esperal implantation have also undergone group therapy, and (b) the success percentage (S + SS) among those receiving group therapy (i.e. the three last therapeutic regimens mentioned in the "gradient" above) amounts to *77 %* as compared to *59 %* in the regimens "aversion treatment" and "disulfiram maintenance treatment" (cf. "therapeutic regimens among all pathological drinkers" in *table 7.4.).* These percentages are *83 %* and *63 %* resp. in the section "therapeutic regimens among alcoholics

& gamma-prealcoholics". *(cf. table 7.4.)* The overall success percentage (S + SS) amounts to *72 %* among alcoholics & gamma-prealcoholics, and *63 %* among all pathological drinkers. The probable reasons for this latter difference were mentioned above, *sub 7.3.1.*

This overall success percentage of *63 %* among all pathological drinkers is near to the *57 %* reported earlier (Wever, 1975); this *63 %* consists of *34 %* "sober" (S) and *29* % "semi-sober" (SS) patients.

Between 1966 and 1969 a total of 291 alcoholics (and other pathological drinkers) have been treated at San Pedro Hospital in Aruba, with an outcome of *25 %* "sober" patients. The present results *seem* to be a little better. However, before 1970 *only* "sobriety" (and not "semisobriety") was considered as successful and no information is available concerning the percentage of "semi-sobriety" in 1966-1969. In that period only "disulfiram maintenance treatment" was employed - of course, after detoxification, treatment of complications etc. Contrary to expectation, therapeutic results were *less* successful among those having undergone aversion treatment (with subsequent disulfiram maintenance) as compared to those on disulfiram maintenance treatment (without previous aversion treatment). This is understandable since a great proportion of those in the latter category ("disulfiram maintenance treatment", cf. *table 7.4.)* were less acutely ill, frequently requiring only outpatient clinic treatment but not hospitalization. For this same reason therapeutic results among those having undergone aversion treatment plus group therapy were also *less* successful than among those on group therapy alone. Therapeutic regimens in other studies have been reported giving succes rates ranging from a minimum of approx. 6 % to a maximum of approx. 80 %, depending on which type of result is considered "successful" (Blaney et al., 1975; Ditman, 1968; Fox, 1968; Ketel, 1963; Poleo Conde, 1971; Rankin, 1969; Santamaria, 1969).

The results in this study therefore may be considered as moderately optimistic, considering the fact that no expert psychiatric help was available in the period studied, whereas this was generally the case in other studies. Hence it can be stated that *treatment of alcoholism* in settings similar to those in this study may be conducted with considerable success by a *general practitioner.*

7.4.2. Disulfiram implantation

Esperal implantation was employed for (a) the "revolving door" alcoholic, or (b) chronic alcoholic - both refusing or "forgetting" to take disulfiram orally. The therapeutic results in this treatment modality are both promising and astonishing:

1) It has a very high success rate only "equalled" by disulfiram maintenance and surpassed by "group therapy" (cf. "therapeutic regimens among alcoholics & gamma-prealcoholics" in *table 7.4.)*, though this effect is diminished by the fact that it also implies the smallest number of patients (20) and the shortest duration of treatment; its success rate is as high as in other reports (Malcolm, 1972; Paillot & Jacques, 1968; Prigent, 1960; Van Erp, 1971);
2) It has a *death rate* (D) *exceeding* the 7 % of the total of 248 patiens, i.e. 15 % (cf. "therapeutic regimens among all pathological drinkers" in *table 7.4.)*.

These *three* deaths included *two chronic alcoholics* and one alcoholic. These deaths are shown in *table 7.9.* (patients no. 30104, 30150, 30196). As shown in *table 7.9.* the following contra-indications for disulfiram have not been adequately taxed among these three patients: (transient) hypertension, convulsive disorders, and psychosis. To this may be added: chronic alcoholism.

Therefore the following recommendations may be given:

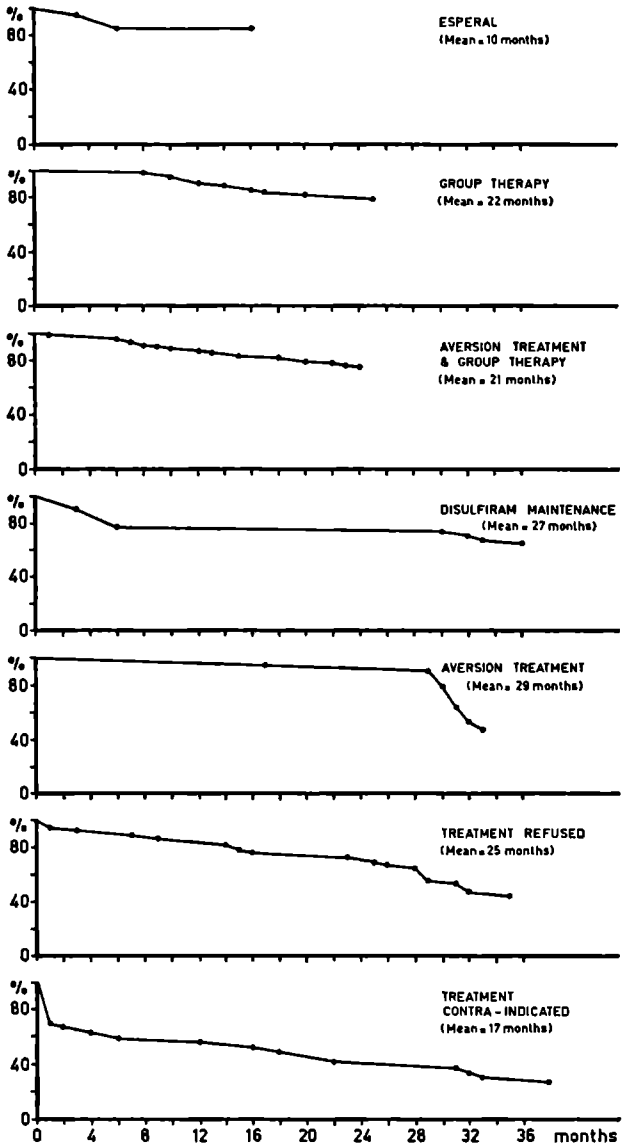
a) *Disulfiram implantation* should *not* be given to *chronic* alcoholics;
b) The *contraindications* for disulfiram have to be observed much *more strictly* in *disulfiram implantation* than in oral disulfiram treatment;
c) *Disulfiram implantation* may be given to *"revolving door"* alcoholics (i.e. frequent recidivism) with possibly greater success than merely oral disulfiram therapy, provided that implantation is preceded by aversion treatment and complemented by group therapy (or other varieties of psychotherapy) - as in this study.

7.4.3. Effect of time lapse on therapeutic results

This effect is shown in *fig. 7.1.* As time goes by, the percentage of "successful" therapeutic results gradually decreases - with the exception of "aversion treatment", in which treatment modality there seems to appear a sharper slope after 29 months. The *slope*

figure 7.1.

EFFECT OF TIME LAPSE ON THERAPEUTIC RESULTS



On the X-axis time is shown in months.
On the Y-axis the percentages are shown of patients who are "sober" (S) or "semi-sober" (SS).

in these decreases in successful treatment results appears to be more or less the same for "Esperal implantation", "group therapy", "aversion treatment plus group therapy" and "disulfiram maintenance treatment" (approx. 6°), while approx. 10° for "treatment refused" and "treatment contraindicated"; for "aversion treatment" approx. 3° before, and approx. 60° after 29 months. A pattern of a negative exponential function (Blaney et al., 1975) may be implied in this slope, except for "aversion treatment". The reason for this sudden decline after 29 months in the "aversion treatment" category is not clear as yet. (Possibly this can be explained by assuming that the group most dependent on individualized treatment (i.e. aversion treatment) by the physician in charge would deteriorate in view of the forthcoming departure of their physician, i.e. the author).

These differences in *slope* are also indicative of the poorer therapeutic effects of the "treatment refused" and "treatment contraindicated" categories at relatively short time lapses, and of "aversion treatment" in the long run.

7.5. CONCLUSIONS

1) Within a relatively short time lapse of three years (1969-1972) the predisposing and perpetuating factors described in Chapters 5 and 6 do *not* give rise to poor therapeutic results. This suggests that the therapeutic regimens used in this study are adequate and able to minimize the effect of those risk factors stimulating the alcoholismic gradient - at least within this short time lapse.
2) Within this short time lapse therapeutic results appear to be influenced positively by therapeutic regimens and AA-membership, and negatively by severe liver damage and diagnostic categories such as chronic alcoholism, problem drinking and gamma-prealcoholism. Among chronic alcoholics this negative effect is mainly due to excessive mortality, and among problem drinkers and gamma-prealcoholics mainly to insufficient motivation to abstinence.
3) The statistically significant effect of "negative relatives' attitudes towards drinker's drinking" and "high versus low AA-involvement" on therapeutic results is *contrary* to expectation. An explanation for this finding is not readily at hand; perhaps

"negative relatives' attitudes" may stimulate the alcoholic in the early years after treatment to strive for abstinence; perhaps "low AA involvement" may stimulate the alcoholic in those early years to look for (more) help from the medical profession. These are, however, merely speculative interpretations.

4) Within a short three years' period a prognosis of therapeutic results on the basis of pre-treatment variables (such as the predisposing and perpetuating factors) is not possible.
5) The high mortality rate (40 % in three years) in chronic alcoholism accentuates the importance of directing the major part of preventive and therapeutic measures towards *non*-chronic alcoholism and its precursor stages instead of chronic alcoholism.
6) The two most valuable contributions to the treatment of alcoholism in Aruba since October 1970 were group therapy and disulfiram-implantation.
7) The overall success rate of 63 % among all 248 pathological drinkers studied is a moderately optimistic one compared to other studies.
8) Treatment of alcoholism in settings similar to those in this study may be conducted with considerable success by a non-specialist general practitioner.
9) Disulfiram implantation is contraindicated in *chronic* alcoholism and *a fortiori* in all those pathological drinkers showing *any* of the contraindications for oral disulfiram.
10) Disulfiram implantation possibly has greater success than oral disulfiram treatment among "revolving door" *non*-chronic alcoholics.
11) There is an apparent gradual decrease in therapeutic results when time goes by among all the therapeutic regimens studied. This decrease is greatest for the "treatment contraindicated", "treatment refused" and "aversion treatment" regimens.

RECOMMENDATIONS

8.1. RECOMMENDATIONS FOR THE PURPOSE OF ESTIMATING THE PREVALENCE AND INCIDENCE OF ALCOHOLISM AND ITS PRECURSOR STAGES

1) The policy of accurate annual registration of the numbers of deaths from (alcoholic) liver cirrhosis should be continued, to enable continuous application of the Jellinek estimation formula, the accuracy of which will increase along with the growth of the population (Chapter 2).
2) The policy of accurate annual registration of import and export of all alcoholic beverages should be continued, and even improved if possible, to enable continuous application of the Ledermann equation (Chapter 2).
3) The results obtained through the population survey method must be considered as having paramount importance for the large scale planning of therapeutic and preventive measures for alcoholism and its precursor stages, in view of the consideration that underestimation is less favorable for these measures than (possible) over-estimation by the survey method.

   The population survey method is also preferable above the Jellinek formula and the Ledermann equation, since it is also able to detect precursor stages, which is not possible trough Jellinek's or Ledermann's method (Chapter 2).
4) The population survey method should be repeated, e.g. with time lapses of five to ten years, in order to determine incidences and prevalences of the diagnostic categories as classified in Chapter 2 (Chapter 2).
5) For the purpose of estimating the maximal prevalence or incidence of chronic alcoholism the population survey method is better suited than the Jellinek formula and the Ledermann equation.

   The prevalence of chronic alcoholism appears to amount to 3 %-10 % (maximally) of the total number of alcoholics.

(Ford, 1956; Fox, 1961; Block, 1962; National Council on Alcoholism, 1970). For the Aruban population (1972) this would imply a maximum of 10 % x 2,530 = 253 chronic alcoholics, with a range of 132 (Ledermann equation: 1,318 alcoholics) to 320 (upper limit of population survey method) (Chapter 2).

8.2. RECOMMENDATIONS FOR THE PURPOSE OF EVALUATING THE EFFECT OF THE PREDISPOSING AND PERPETUATING FACTORS ON THE ALCOHOLISMIC GRADIENT

The population survey method should be repeated, e.g. with time lapses of five to ten years, within the sample studied in 1972, in order to determine whether the predisposing and perpetuating factors have provoked shifts on the alcoholismic gradient or not. These shifts have been observed in the U.S.A. (Cahalan & Cisin, 1976), though in both directions (Chapters 2, 5 & 6).

8.3. RECOMMENDATIONS FOR THE PURPOSE OF EARLY DETECTION OF ALCOHOLISM AND ITS PRECURSOR STAGES ESPECIALLY IN GENERAL PRACTICE

1) Whenever alcoholism or its precursor stages are suspected in a patient, an alternative approach may be to start with an evaluation of predisposing and perpetuating factors (as described in Chapters 4, 5 and 6) in that patient.
This is due to the fact that alcoholism is characterized by the patient's denial of questions directly related to excessive drinking. Such an evaluation of the risk factors mentioned above may fortify the suspected diagnose, e.g. if 50 % of the risk factors evaluated are found positive. After this evaluation, questions may be asked directly related to the diagnoses of alcoholism or its precursor stages as described in Chapter 2, either to the patient himself or to his wife, children, parents or other significant relations. After both these inquiries (a. evaluation of risk factors; b. diagnostic questions) the rank order of procedures must imply: physical examination, additional laboratory, and if necessary radio-

logic and other procedures, such as ECG; these are mainly directed to discover alcoholismic and other complications like those described in Chapter 7.

2) Early detection of risk factors (especially predisposing factors) should be recommendable especially for *males younger than 30 years* and for *females,* since in these subdivisions the best correlations were found between the alcoholismic gradient and these risk factors.

8.4. RECOMMENDATIONS FOR THE PURPOSE OF IMPROVING TREATMENT RESULTS IN ALCOHOLICS AND OTHER PATHOLOGICAL DRINKERS

1) Group therapy as described in Chapter 7 should be continued and if possible improved through e.g. participation of qualified group psychotherapist, psychiatrists and other experts in this field (Chapter 7).
2) Disulfiram implantation, preceded by aversion treatment and disulfiram and group therapy, should be continued with *strict* observations of indications and contraindications (Chapter 7).
3) Participation of Alcoholics Anonymous (AA), Al-Anon and Alateen in group therapy should be intensified.
4) Participation of wives, husbands, children, parents and siblings of alcoholics in group therapy should be intensified.
5) Methods should be sought to better motivate *problem drinkers* and other *prealcoholics* towards accepting treatment. Joining Alcoholics Anonymous, Al-Anon and Alateen can be most valuable for this purpose.

8.5. RECOMMENDATIONS FOR THE PURPOSE OF PREVENTING FUTURE ALCOHOLISM

1) The early identification in general practice of "high risk" families (i.e. those scoring high for the predisposing and perpetuating factors as described in Chapters 4, 5 and 6) should be viewed as an important contribution to prevention especially with regard to the children in such families. In this sense it can be stated, that early identification and

treatment of alcoholic parents, siblings and other relatives implies excellent prevention of the alcoholismic gradient in the children in such families.

2) Education of the population at large should be considered - e.g. through school education and mass media, family physicians and other helpers - away from those predisposing and perpetuating factors which presumably can be changed: *e.g. paternal and maternal inebriety; inebriety in siblings and children; severe interparental and parent-child conflicts; unfavorable paternal examples (P.B.I.-factors); marital conflicts; social pressure to drink and hospitality drinking; tolerant attitudes towards (excessive) drinking; violation of confidence; authority conflicts with parents; oral fixation.*
3) Alcoholism and its precursor stages should be viewed in the broad context of all these predisposing and perpetuating factors, and a conjoint approach should be propagated of all these pathological complementary relationships (cf. Chapter 4) as a whole.
4) Even more important is to indoctrinate the population at large, that *alcohol* is not the only means to escape from stressful life events like many of the predisposing and perpetuating factors, but that better solutions can be found through consultation of the family physician and other helpers.
5) Improvement of such single factors (e.g. poor housing conditions, unemployment, anxiety, suicidality, boredom, introversion, drug dependence, and dissatisfaction with one's health, housing or achievement) through help from social workers (to find better housing, employment, recreational facilities to combat boredom), or from physicians (to treat anxiety, depression and suicidality, introversion, dissatisfaction, or drug dependence), will most probably help to prevent recidivism or progression on the alcoholismic gradient.
6) It should be seriously considered to increase the price of beverage alcohol relative to mean per capita income at a rate which would percentually exceed annual increase in

income, since - in agreement with other studies - it was shown in Chapter 5 that "economic availability" stimulates the alcoholismic gradient.

8.6. RECOMMENDATIONS FOR THE PURPOSE OF PLANNING THERAPEUTIC AND PREVENTIVE MEASURES FOR ALCOHOLICS, CHRONIC ALCOHOLICS AND OTHER PATHOLOGICAL DRINKERS

1) Alcoholics in the general hospital can be treated optimally in a special unit, in order to prevent them from being morally undermined by other patients (Ehik, 1968; Esser, 1965).
   In the general hospital there should be space for group therapy, AA meetings and information services.
2) Since the prevalence of *chronic* alcoholism is estimated to be 253 (range 132-320) in Aruba in 1972, as compared to 2,530 alcoholics (alcoholics with and without complications *plus* chronic alcoholics) it is highly recommendable to invest more energy and finances in the planning of the care for *non*-chronic alcoholics.
   This view is supported by the fact that in this study chronic alcoholics showed a mortality rate of 40 % over three years: they tend to eliminate themselves rapidly through excessive mortality. Moreover in chronic alcoholism generally there is increasing brain damage (various forms of dementia) which progressively impedes the individual's capacity to perceive the importance of sobriety.
   The Aruban "Foundation against Alcohol Abuse" ("Stichting Bestrijding Alcohol Misbruik Aruba") is since the nineteen-sixties engaged in planning a Rehabilitation Center/Halfway House for *chronic* alcoholics - a center which intends to admit approx. 30 chronic alcoholics per trimester.
   At a number of 253 chronic alcoholics (1972) this Rehabilitation Center would finish its task in two or maximally three years. That is, disregarding the recidivism rate of chronic alcoholics.
3) It should be highly recommended, to devise this Rehabilitation Center not only for the treatment and rehabilitation

of chronic alcoholics, but also for alcoholics, (gamma-)pre-alcoholics and problem drinkers, since in such a setting this would imply meaningful work for many decades, simply because the latter categories are much more numerous.

At the same time such an approach would mean substantial relief for the island's only hospital where from 1969 through 1972 there were 100-120 annual admissions for pathological drinking, while average stay in hospital was 2-3 weeks, and while daily 5-10 hospital beds were occupied by these patients.

4) The Rehabilitation Center as described above may also be used as a center for the treatment of drug dependence or addiction.

   Alcoholism and drug addiction are closely related, have analogous treatment modalities, and one thus achieves a coordinated approach to a number of diseases with marked resembances from the point of view of symptomatology, social-economic consequences, psychosocial implications and possibly of etiology.

5) The Rehabilitation Center should contain a minimum of 10 beds, 1 isolation cell, 1 room for group therapy and AA meetings, and 2 consultation rooms as an outpatient clinic. It should be headed by 1 psychiatrist, 1 general practitioner, 1 internist (all part-time), 2 social workers, 3 AA officers, 1-2 secretaries and a sufficient number of nurses (full-time).

6) It may be suggested to devise this Rehabilitation Center also as a center for scientific research in alcoholism and drug dependence. This research should embody:
   a) studies of predisposing and perpetuating factors as done in this study;
   b) studies of somatic complications of alcoholism and drug dependence;
   c) studies of mental complications;
   d) biochemical aspects of e.g. physical dependence, tolerance, liver damage;
   e) hematological aspects of alcoholism or drug dependence;
   f) factors affecting prognosis;
   g) studies of various therapeutic regimens.

SUMMARY

Chapter 1 gives a description of the purposes of this study: estimation of the prevalences of alcoholism and its precursor stages, i.e. the alcoholismic gradient; estimation of the prevalences of a number of (socio-)medical, social and psychological characteristics of the alcoholismic gradient, evaluation of a number of (socio-) medical, social and psychological factors, as regards their contribution to the etiology of alcoholism; evaluation of therapeutic results and factors possibly affecting prognosis.

Moreover, in this chapter definitions of concepts important in this study, a global review of applicated methods, and a description of the population samples studied, are given.

In *Chapter 2,* after a description of the methods employed, prevalences are estimated of alcoholism, gamma-prealcoholism, prealcoholism, problem drinking, social drinking and abstinence in Aruba.

Factor analysis did confirm the unidimensional, but not the multidimensional models of "alcoholism" and "problem drinking" for the Aruban population.

The results with the three estimation methods employed, show significant differences, though in the same order of magnitude; a number of explanations for this observation are proposed.

For estimating the prevalence of chronic alcoholism, the results show smaller variations.

In *Chapter 3* differences are mentioned among the categories classified in Chapter 2, concerning: frequencies of alcohol consumption and intoxication, mean daily alcohol consumption, monthly expenses on beverage alcohol, beverage preference, average duration of drinking bouts, excessive drinking in weekends and at other culturally determined occasions, Jellinek's phaseology, tolerance, age distribution, and certain public opinions concerning alcoholics and AA (Alcoholics Anonymous).

Chapter 4 gives a theoretical orientation in those theories concerning the etiology of alcoholism and its precursor stages, which are considered important in this study.

The difference between "predisposing" and "perpetuating" factors in the etiology of alcoholism is explained. These factors can be considered as risk factors for alcoholism.

In *Chapter 5*, after a description of the methods employed, the results are presented of the prevalences of "predisposing" and "perpetuating" factors, among the categories classified in Chapter 2. These prevalences were determined among: (a) female respondents; (b) male respondents younger than 30 years; (c) male respondents older than 30 years; (d) all respondents regardless of sex and age. In *Chapter 6* a conjoint evaluation is presented of all significant "predisposing" factors separately, and all significant "predisposing" plus "perpetuating" factors, among the categories classified in Chapter 2.

The hypothesis, that an increase in numbers of risk factors is significantly correlated with an increase on the alcoholismic gradient, could be convincingly confirmed.

For males younger than 30 years "predisposing" factors seem to play a relatively more important role in the etiology of alcoholism, than for males older than 30 years.

For males older than 30 years "perpetuating" factors seem to play a relatively more important role in the perpetuation of alcoholism, than for males younger than 30 years; for female respondents of every age and for the total group of male and female respondents (N = 224), the same observation is applicable as for males older than 30 years.

Discriminant function analysis of the risk factors seems to have an important predictive capacity for the adequate classification of the diagnostic categories: 70 %—100 % of respondents are adequately classified through this method.

Chapter 7 gives a review of the 248 pathological drinkers treated, diagnoses, policlinical and clinical evaluation and treatment procedures, and the ways of cooperation with AA (Alcoholics Anonymous).

Results are presented of an investigation of the possible influence of a number of factors on therapeutic outcome as well as therapeutic results in 7 therapeutic regimens. It appears, that within a relatively short period (1969-1972) "predisposing" and "perpetuating" factors do not give rise to poorer therapeutic results.

Therapeutic results within such a short period appear to be favorably influenced by the kind of therapeutic regimen and AA-membership and unfavorably by severe liver damage and certain varieties of pathological drinking such as chronic alcoholism, problem

drinking and gamma-prealcoholism. Successful treatment results were attained in 63 % of 248 pathological drinkers.

Chapter 8 gives a set of recommendations, directly deduceable from the findings in the preceding chapters, concerning diagnosis, prevalence estimation, prevention, treatment and research in the future.

In final consideration of the design and results of this retrospective study, it is essential, to mention that this study has a number of limitations:

— since its findings are only applicable to the small and heterogeneous population of Aruba;
— since the reliability of answers to questions in questionnaires is limited, while the basis of this study is an inquiry, which implies a methodological weakness;
— since a number of criteria mentioned in Chapter 2, can be considered as relatively "weak" i.e. exclusively anamnestic data (e.g. alcoholic "gastritis" or "polyneuropathy"), in contrast to the relatively "hard" data, regarding 248 patients mentioned in Chapter 7, and which imply both medical history and clinical examination;
— since the classification in diagnostic categories (Chapter 2) like in other surveys on alcoholism (Cahalan et al., 1969; Selzer et al., 1975) implies an arbitrary choice of the investigator, which means that there probably is no stringent or complete congruency with these categories in reality.

It is also essential to point to the differences in age and sex distribution, between the experimental group of pathological drinkers (survey C) and the control group (survey B).

In an attempt to eliminate the influence of age and sex, it was decided to subdivide this combined group (survey B & C) in: female respondents, male respondents younger than 30 years; male respondents older than 30 years. (Chapter 5).

The (quasi-) mathematical approach of the alcoholismic gradient in this study should be viewed in the perspective of "the elusive nature" of this problem (De Lint, 1974): a definitive grip on the problem has remained impossible throughout this study, though the impression prevails that the problem has been approached satisfactorily.

Hoofdstuk 1 geeft een beschrijving van de doelstellingen van dit onderzoek: het schatten van de prevalenties van alcoholisme en voorstadia daarvan, d.w.z. de alcoholismische gradiënt; het bepalen van de prevalenties van een aantal (sociaal-)medische, sociale en psychologische karakteristieken van de alcoholismische gradiënt; het toetsen van een aantal (sociaal-)medische, sociale en psychologische factoren m.b.t. hun bijdrage aan de aetiologie van alcoholisme; het evalueren van therapieresultaten en van factoren die de prognose mogelijkerwijs beïnvloeden. Tevens worden in dit hoofdstuk de definities van voor dit onderzoek belangrijke begrippen, een globaal overzicht van de gebruikte methoden, en een beschrijving van de onderzochte populaties gegeven.

In *Hoofdstuk 2* wordt, voorafgegaan door een beschrijving van de gebruikte methoden, een schatting gemaakt van de prevalenties van alcoholisme, gamma-prealcoholisme, prealcoholisme, probleemdrinken, sociaal drinken en geheelonthouding op Aruba. Een eventuele voorkeur voor multidimensionale modellen van "alcoholisme" en "probleemdrinken" boven unidimensionale, wordt op grond van factor analyse in dit onderzoek voor de Arubaanse populatie *niet* bevestigd. De resultaten met de drie gebruikte schattingsmethoden vertonen - hoewel in dezelfde orde van grootte - belangrijke verschillen; hiervoor worden enige verklaringen gegeven. Voor een schatting van de prevalentie van *chronisch* alcoholisme is de spreiding in de uitkomsten minder groot.

In *Hoofdstuk 3* worden voor de in Hoofdstuk 2 geclassificeerde categorieën de gevonden verschillen vermeld betreffende o.a.: frequenties van drinken en dronkenschap, gemiddelde dagelijkse alcoholconsumptie, maandelijkse uitgaven aan alcoholhoudende drank, drankvoorkeur, gemiddelde tijdsduur van "drinking bouts", excessief drinken in weekends en op andere cultureel bepaalde dagen, Jellinek's phaseologie van het drinken, tolerantie, leeftijdsverdeling, en bepaalde publieke opvattingen t.a.v. alcoholisten en de Anonieme Alcoholisten (AA).

Hoofdstuk 4 geeft een theoretische oriëntatie in de voor dit onderzoek belangrijke theorieën betreffende de aetiologie van alcoholisme en voorstadia daarvan. Het verschil tussen "predisponerende" en

"perpetuerende" factoren in deze aetiologie wordt nader uiteengezet. Deze factoren kunnen als risicofactoren tot alcoholisme beschouwd worden.

In *Hoofdstuk 5* worden, voorafgegaan door een beschrijving van de gebruikte methoden, de resultaten vermeld van de prevalenties van "predisponerende" en "perpetuerende" factoren in de in Hoofdstuk 2 geclassificeerde categorieën. Deze prevalenties zijn bepaald onder: (a) vrouwelijke respondenten; (b) mannelijke respondenten jonger dan 30 jaar; (c) mannelijke respondenten ouder dan 30 jaar; (d) alle respondenten ongeacht geslacht of leeftijd.

In *Hoofdstuk 6* wordt een gezamenlijke evaluatie gegeven van alle significante "predisponerende" factoren afzonderlijk en van alle significante "predisponerende" plus "perpetuerende" factoren in de in Hoofdstuk 2 geclassificeerde categorieën. De hypothese, dat een toename van het aantal risicofactoren significant correleert met een progressief opschuiven op de gradiënt: abstinentie → sociaal drinken → probleemdrinken → (gamma-)prealcoholisme → alcoholisme, kon overtuigend bevestigd worden.

Voor mannen jonger dan 30 jaar lijken "predisponerende" factoren een relatief grotere rol te spelen in de aetiologie van alcoholisme dan voor mannen ouder dan 30 jaar. Voor mannen ouder dan 30 jaar lijken "perpetuerende" factoren een relatief grotere rol te spelen bij het in stand houden van alcoholisme dan voor mannen jonger dan 30 jaar; voor vrouwelijke respondenten (ongeacht leeftijd) en voor de totale groep van mannelijke en vrouwelijke respondenten (N = 224) geldt hetzelfde als voor mannen ouder dan 30 jaar.

"Discriminant function analysis" van de onderzochte risicofactoren lijkt een belangrijk voorspellend vermogen te hebben voor het juist classificeren van de diagnostische categorieën : 70 %-100 % der respondenten wordt hiermee goed ingedeeld.

Hoofdstuk 7 geeft een overzicht van de 248 behandelde patiënten met alcoholisme of voorstadia daarvan, gestelde diagnoses, poliklinische en klinische evaluatie- en behandelingsprocedures, en van de wijze van samenwerken met de AA (Alcoholics Anonymous). Tevens worden de resultaten vermeld van een onderzoek naar de mogelijke invloed van een aantal factoren op behandelingsresultaten, alsmede de behandelingsresultaten onder 7 therapeutische regimes besproken. Het blijkt, dat binnen een relatief korte periode (1969-1972) de

"predisponerende" en "perpetuerende" factoren *géén* aanleiding geven tot slechtere behandelingsresultaten. Therapeutische resultaten blijken binnen een dergelijke korte periode *gunstig* beïnvloed te worden door de aard van het therapeutisch regime en AA-lidmaatschap, - en *ongunstig* door ernstige leverbeschadiging en bepaalde vormen van pathologisch drinken zoals chronisch alcoholisme, probleemdrinken en gamma-prealcoholisme.

De behandelingsresultaten waren succesvol onder 63 % van de 248 behandelde "pathologische drinkers".

Hoofdstuk 8 geeft een aantal aanbevelingen, direct herleidbaar uit de bevindingen in de voorgaande hoofdstukken, t.a.v. diagnostiek, schatting van prevalenties, preventie, therapie en research in de toekomst.

Bij een nabeschouwing van de opzet en resultaten van dit retrospectief onderzoek is het zinvol, te vermelden, dat dit onderzoek een aantal beperkingen heeft, o.a.:

— doordat het beperkt is tot de kleine, heterogene bevolking van Aruba;
— doordat de betrouwbaarheid van het beantwoorden van vragen in enquêtes beperkt is, terwijl juist de basis van dit onderzoek een mondelinge enquête is, hetgeen derhalve een methodologische zwakte impliceert;
— doordat een aantal criteria, zoals vermeld in Hoofdstuk 2, "zachte" d.w.z. uitsluitend anamnestische gegevens zijn (b.v. alcohol "gastritis", alcohol "polyneuritis"), in tegenstelling tot de "hardere" in Hoofdstuk 7 vermelde gegevens van 248 patiënten, welke berusten op anamnese plus klinisch onderzoek;
— doordat de classificatie in diagnostische categorieën (Hoofdstuk 2) - zoals ook in andere enquêtes over alcoholisme (Cahalan et al., 1969; Selzer et al., 1975) - een arbitraire keuze van de onderzoeker impliceert, en niet volledig congruent behoeft te zijn met deze categorieën in de realiteit.

Het is eveneens zinvol te wijzen op de geconstateerde verschillen in leeftijds- en geslachtsverdeling tussen de experimentele groep van pathologische drinkers *(survey C)* en de controlegroep *(survey B)*. Om de invloed van leeftijd en geslacht zoveel mogelijk te elimineren

werd daarom besloten om deze gezamenlijke groep *(survey B & C)* onder te verdelen in: vrouwelijke respondenten; mannelijke respondenten jonger dan 30 jaar; mannelijke respondenten ouder dan 30 jaar (Hoofdstuk 5).

De (quasi-)mathematische benadering van de alcoholismische gradiënt in dit onderzoek dient beschouwd te worden tegen de achtergrond van het "elusieve (ontwijkende) karakter" van deze problematiek (De Lint, 1974): het definitief "grijpen" van het probleem is in iedere phase van dit onderzoek onmogelijk gebleven, hoewel de indruk bestaat dat het probleem adequaat benaderd is.

Aitchison, J & Brown, J A C (1966) The lognormal distribution Cambridge University Press, Cambridge

Albrecht, G L (1973) The alcoholism process a social learning viewpoint p 11—42 In P G Bourne & R Fox (1973) Alcoholism Progress in Research and Treatment Academic Press Inc, New York 439 + XIV pp

Amark, C (1951) A study in alcoholism Clinical, social-psychiatric and genetic investigations Acta psychiat neurol Scand Suppl 70

American Psychiatric Association (1968) Diagnostic and statistical manual of mental disorders

Argeriou, M (1974) The Jellinek estimation formula revisited Quarterly Journal of Studies on Alcohol *35*, 1053 1057

Arntzenius, A C, de Haas, J H & Styblo K (1976) CB Heart Project in the Netherlands Grouping of risk factors Hart Bulletin *7*, 75—81

Artzenius, A C & Styblo, K (1977) CB Heart Project in the Netherlands Critical discussion of one year intervention results Hart Bulletin *8*, 60—65

Bacon, M K (1973) Cross-cultural studies of drinking p 171—194 In P G Bourne & R Fox (1973) Alcoholism Progress in Research and Treatment Academic Press Inc, New York, 439 +XIV pp

Bailey, M B (1961) Alcoholism and marriage A review of research and professional literature Quarterly Journal of Studies on Alcohol *22*, 81—97

Bailey, M & Leach, B (1965) Alcoholics Anonymous, pathway to recovery A study of 1,058 members of the AA fellowship in New York City National Council on Alcoholism Inc, New York 58 + IV pp

Bailey, M B & Stewart, J (1967) Normal drinking by persons reporting previous problem drinking Quarterly Journal of Studies on Alcohol *28*, 305—315

Bales, R F (1962) Attitudes toward drinking in the Irish culture p 157—187 In D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley, London 616 + XVIII pp

Beaubrun, M H (1971) Socio-cultural factors affecting the incidence and treatment of alcoholism in the Caribbean Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 122—134 Verenigde Antilliaanse Drukkerijen, Aruba

Beaubrun, M H (1969) Alcoholism treatment in Trinidad and Tobago A description and review of a ten year treatment programme 29 pp Proceedings of Workshop on Alcoholism, U S Virgin Islands

Bell, R A, Keeley, K A, Clements, R D, Warheit, G J & Holzer, C E (1976) Alcoholism, life events and psychiatric impairment p 467—480 In F A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 467-480

Berkley, L (1956) Het alcoholprobleem op Aruba Scriptie Sociale Academie, Findhoven

Berkley, L (1957) The alcohol problem in Aruba p 50—65 In Constructive mental hygiene in the Caribbean (1957) Van Gorcum, Assen XIV + 176 pp

Blaney, R, Radford, I S & MacKenzie, G (1975) A Belfast study of outcome in the treatment of alcoholism British Journal of Addiction *70*, 41—50

Block, M A (1962) Alcoholism, its facets and phases The John Day Company, New York 320 pp

Bosch, G B (1836) Reizen in West-Indie Deel II Utrecht

Braat, J P (1957) p 18—20 In Constructive mental hygiene in the Caribbean (1957) Van Gorcum, Assen XIV +176 pp

Brenner, B (1959) Estimating the prevalence of alcoholism, toward a modification of the Jellinek formula Quarterly Journal of Studies on Alcohol *20*, 255—260

Bruun, K, Edwards, G, Lumio, M, Makela, K, Pan, L, Popham, R E, Room, R, Schmidt, W, Skog O J, Sulkunen, P & Österberg, E (1975) Alcohol control policies in public health perspective The Finnish Foundation for Alcohol Studies, Vol 25 106 pp

Bijl, O A (1971) Message from the Public Health Department Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 14 15 Verenigde Antilliaanse Drukkerijen, Aruba

Cadoret, R J (1976) Genetic determinants of alcoholism p 225—256 In R E Tarter & A A Sugerman (1976) Alcoholism Interdisciplinary Approaches to an Enduring Problem Addison Wesley Publishing Company, Inc Advanced Book program Reading, Massachusetts, U S A 857 + XVI pp

Cahalan, D & Cisin, I H (1968) American drinking practices summary of findings from a national probability sample I Extent of drinking by population subgroups Quarterly Journal of Studies on Alcohol *29*, 130—152

Cahalan, D, Cisin, I H & Crossley H M (1969) American Drinking Practices A National Study of Drinking Behavior and Attitudes Publications Division, Rutgers Center of Alcohol Studies New Brunswick, New Jersey, U S A College & University Press New Haven, Connecticut, U S A 260 + XXVI pp

Cahalan, D & Cisin, I H (1976) Epidemiological and social factors associated with drinking problems p 523—572 In R E Tarter & A A Sugerman (1976) Alcoholism Interdisciplinary Approaches to an Enduring Problem Addison Wesley Publishing Company, Inc Advanced Book Program Reading, Massachusetts, U S A 857 + XVI pp

Camps, F E & Dodd, B E (1967) Increase in the incidence of non-secretors of ABH blood group substances among alcoholic patients Br med J *1*, 30

Canavan, D (1971) A doctor talks to alcoholic patients Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 41—51 Verenigde Antilliaanse Drukkerijen, Aruba

Caravedo, B (1957) Acculturation problems in Latin America p 85 87 In Constructive mental hygiene in the Caribbean (1957) Van Gorcum, Assen XIV + 176 pp

Chu, G (1972) Drinking patterns and attitudes of rooming-house Chinese in San Francisco Quarterly Journal of Studies on Alcohol, Supplement No 6, 58—68

Compendium Gezondheidsstatistiek Nederland 1974 (1974) Central Bureau of Statistics, Ministry of Public Health and Environmental Hygiene Staatsuitgeverij, The Hague 371 + X pp

Conger, J H (1956) Reinforcement theory and the dynamics of alcoholism Quarterly Journal of Studies on Alcohol *17*, 296—305

Cork, R M (1969) The Forgotten Children A Study of Children with Alcoholic Parents PaperJacks, a division of General Publishing Co Ltd, Don Mills, Canada 112 pp

Criteria Committee, National Council on Alcoholism (1972) Criteria for the diagnosis of alcoholism Annals of Internal Medicine *77*, 249—258

Croes, H S (1971) Some juridical aspects of alcoholism in Aruba Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 118-121 Verenigde Antilliaanse Drukkerijen, Aruba

Cruz Coke, R & Varela, A (1966) Inheritance of alcoholism Its association with colour blindness Lancet *2*, 1282—1284

Dahlgren, L (1975) Special problems in female alcoholism British Journal on Addiction *70* Suppl Conference Alcoholism, 18—24

Dalhuysen, B (1971) Internal medical aspects of alcoholism Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 100-105 Verenigde Antilliaanse Drukkerijen, Aruba

Davies, D L (1962) Normal drinking in recovered alcohol addicts Quarterly Journal of Studies on Alcohol *23*, 94—104

Davies, D L, Scott, D F & Malherbe, M E L (1969) Resumed normal drinking in recovered psychotic alcoholics International Journal of the Addictions *4* (2), 187—194
DECO, Dienst Economische Ontwikkeling Aruba (1970) Notitie over het verbruik van alcoholhoudende dranken op Aruba
DECO, Dienst Economische Ontwikkeling Aruba (1970a) Vier jaar alcoholmisbruikbestrijding op Aruba, 1966 t/m 1969
DECO, Dienst Economische Ontwikkeling Aruba (1971) Voor verbruik beschikbaar gekomen hoeveelheden alcoholische dranken per jaar en per hoofd van de bevolking op het eilandgebied Aruba
De Haas, J H (1976) CB-Project Risicofactoren Hartziekten Samenvattende beschouwing over eerste uitkomsten Hart Bulletin *7*, 38—41
De Haas, J H (1977) CB Project Risicofactoren Hartziekten Samenvattende beschouwing over resultaten van interventie Hart Bulletin *8*, 35—41
De Lint, J (1968) Alcohol use in Canadian society Addictions *14*, 14-28
De Lint, J (1974) The epidemiology of alcoholism the elusive nature of the problem, estimating the prevalence of excessive alcohol use and alcohol-related mortality, current trends and the issue of prevention p 75—108 In N Kessel, A Hawker & H Chalke (1974) A Medical Profile, Proceedings of the First International Medical Conference on Alcoholism Edsall & Co, London
De Lint, J (1975) Estimating rates of excessive alcohol use and alcohol-related health damage Proceedings of the 21st International Institute on the Prevention and Treatment of Alcoholism, Helsinki
De Lint, J (1975a) Current trends in the prevalence of excessive alcohol use and alcohol-related health damage British Journal on Addiction *70*, 3—13
De Lint, J & Schmidt, W (1968) The distribution of alcohol consumption in Ontario Quarterly Journal of Studies on Alcohol *29*, 968—973
De Lint, J & Schmidt, W (1970) Consumption averages and alcoholism prevalence a brief review of epidemiological investigations Proceedings of the 16th International Institute on the Prevention and Treatment of Alcoholism, Lausanne
De Lint, J & Schmidt, W (1970a) Mortality from liver cirrhosis and other causes in alcoholics A follow-up study of patients with and without a history of enlarged fatty liver Quarterly Journal of Studies on Alcohol *31*, 705—710
De Lint, J & Schmidt, W (1971) Drinking habits and public health, some observations Proceedings of the 17th International Institute on the Prevention and Treatment of Alcoholism, West Berlin
De Lint, J & Schmidt, W (1971a) Consumption averages and alcoholism prevalence a brief review of epidemiological investigations Br J Addict *6*, 97—107
De Lint, J & Schmidt, W (1971b) Alcohol use and alcoholism Addictions *18*, 1—15
De ontwikkeling van het toerisme in de Nederlandse Antillen van 1964 t/m 1974 (1974) Bureau voor de Statistiek Nederlandse Antillen, Departement Sociale en Economische Zaken, Curaçao, Netherlands Antilles 17 pp
De Vries, A E N (1973) Gezin en hulpverlening in dorpbijstad Dekker & van de Vegt, Nijmegen, The Netherlands 188 + X pp
De Windt, H L (1971) Neuropsychiatric aspects of alcoholism in the Netherlands Antilles, especially referring to Aruba Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 91—95 Verenigde Antilliaanse Drukkerijen, Aruba
Diem, K & Lenther, C (1970) Scientific tables Ciba-Geigy Ltd, Basel 810 pp
Ditman, K S (1968) "Do we have a specific treatment for alcoholism?" Int J Psychiat *5*, 48—52
Dozier, E P (1966) Problem drinking among American Indians The role of sociocultural deprivation Quarterly Journal of Studies on Alcohol *27*, 72-87.

Driessen, W C H (1971) Alcoholism, guilt and disease Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 196—200 Verenigde Antilliaanse Drukkerijen, Aruba
Edwards, G (1970) The status of alcoholism as a disease p 140—163 In R V Phillipson (1970) Modern Trends in Drug Dependence and Alcoholism Butterworths, London
Eerste algemene volks en woningtelling Nederlandse Antillen (1972) Bureau voor de Statistiek, Departement Sociale en Economische Zaken, Curaçao, Netherlands Antilles
Efron V & Keller, M (1970) Selected statistics on consumption of alcohol and alcoholism (1930—1968) Publications Division, Rutgers Center of Alcohol Studies, New Brunswick, U S A 18 pp
Ehik, J (1968) The use of thioridazine in the treatment of acute brain syndrome due to alcohol Journal of the Medical Association of Georgia *57*, 347—348
Eicher, E R (1973) The hematologic disorders of alcoholism American Journal of Medicine, *54*, 621—630
Encel, S, Kotowicz, K C & Resler, H E (1972) Drinking patterns in Sydney, Australia Quarterly Journal of Studies on Alcohol, Supplement No 6, 1—27
Erkelens, D W (1975) The influence of alcohol and carbohydrates on hypertriglyceridaemia Thesis, Groningen 111 pp
Erkelens, D W (1976) Alcohol en vetstofwisseling T Alc Drugs *2*, 14—18
Esser, P H (1965) Alcoholism in the Netherlands Psychiatria, Neurologia, Neurochirurgia *68*, 371—386
Esser, P H (1967) Klinische psychiatrie J H Kok N V, Kampen, The Netherlands 526 pp
Fine, E W, Yudin, L W, Holmes, J & Heinemann, S (1976) Behavioral disorders in children with parental alcoholism p 507—517 In F A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 507—517
Ford, L C (1956) The general practitioner's role in alcoholism The Linacre Quarterly 14 pp
Fox, R (1961) The alcoholics in a general hospital In L Linn Frontiers in general hospital psychiatry International Universities Press 14 pp
Fox, R (1967) A multidisciplinary approach to the treatment of alcoholism Amer J Psychiat *123*, 769—778
Fox, R (1968) A multidisciplinary approach to the treatment of alcoholism Int J Psychiat *5*, 34—44
Fox, R (1971) A multidisciplinary approach to alcoholism Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 29—36 Verenigde Antilliaanse Drukkerijen, Aruba
Friedman, M, Hatcher, G & Watson, L (1967) Primary hypomagnesaemia with secondary hypocalcaemia in an infant Lancet *1*, 703—705
Gadourek, I (1963) Riskante gewoonten en zorg voor eigen welzijn Wolters, Groningen 516 pp
Gant, J C (1968) Treatment of the alcohol withdrawal syndrome Journal of the Tennessee Medical Association *61*, 45—47
Gerrits, W B J & Vandenbroucke, J (1971) Leverziekten, ziekten van galwegen en pancreas p 177—200 In L B W Jongkees & J Vandenbroucke (1971). Codex Medicus Agon Elsevier, Amsterdam/Brussels 1552 + XIII pp
Gips, C H (1975) Alkoholgebruik 1875—1974 en sterfte door levercirrose in Nederland T Alc Drugs *1*, 24—29
Gips, C H (1976) Recente gegevens over het alkoholgebruik en over de sterfte aan levercirrose in Nederland T Alc Drugs *2*, 131—133
Glatt, M M (1975) Today's enjoyment-tomorrow's dependency The road towards the "rock bottom" and the way back British Journal on Addiction *70*, Suppl Conference on Alcoholism, 25—34

Gomberg, E S (1976) The female alcoholic p 603—636 In R E Tarter & A A Sugerman (1976) Alcoholism Interdisciplinary Approaches to an Enduring Problem Addison-Wesley Publishing Company, Inc Advanced Book Program Reading, Massachusetts, U S A 857 +XVI pp
Gorrell, G J (1967) Inheritance of alcoholism Lancet *1*, 274
Hartog, J (1953) Aruba, zoals het was, zoals het werd Gebroeders De Wit, Aruba 498 + XVI pp
Heath, D B (1962) Drinking patterns of the Bolivian Camba p 22—36 In D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley London 616 + XVIII pp
Henríquez, N E (1955) Amigoe di Curacao, October 14, 1955
Heydendael, P H J M, Huygen, F J A, Ketelaar-van Iersel, A M J, Persoon, J M G, Windhausen-Mertens, M, Van Thiel, A & Vissers, A (1972) Gezin en ziekte Een verkennend onderzoek naar psychosociale en medische gezinsfaktoren en hun onderlinge samenhang Dekker & Van de Vegt, Nijmegen 204 + VIII pp
Hoetink, H (1958) Het patroon van de oude Curacaose samenleving Een sociologische studie Van Gorcum, Assen, The Netherlands 187 + VIII pp
Hoff, E C (1967) Motivating the alcoholic to accept rehabilitative therapy p 241—256 In R P Maickel (1967) Biochemical Factors in Alcoholism Pergamon Press, Oxford 256 + XI pp
Holzgreve, W (1970) Frauenalkoholismus — seine Ursachen, Auswirkungen und Behandlungsmoglichkeiten p 11—15 In R Battegay et al (1970) Alkoholismus bei Frauen Hohenecck Verlag GmbH, Hamm, Germany 96 pp
Hore, B D & Wilkins, R H (1976) A general-practice study of the commonest presenting symptoms of alcoholism Journal of the Royal College of General Practitioners *26*, 140—142
Horton, D (1943) The functions of alcohol in primitive societies a cross-cultural study Quarterly Journal of Studies on Alcohol *4*, 199—320
Horwitz, J, Marconi, J & Castro, G A (1967) Epidemiología del alcoholismo en América Latina Acta, Buenos Aires 206 pp
Hrynkiewicz, L, Cyganik, L & Luszpinski, J (1966) Ocena wlasnych doswiadczen w stosowaniu implantacji esperalu u alkoholikow (Evaluation of personal experiences in treatment of alcoholics by Esperal implantation) Probl Alkzmu, Wars *14*, 7—8
Hyland, J & Scott, S (1969) Alcohol consumption tables an application of the Ledermann equation to a wide range of consumption averages Addiction Research Foundation, Toronto, Canada
Isbell, H (1970) Clinical characteristics and relationships of different types of alcohol and drug dependence Proceedings of 29th International Congress on Alcoholism and Drug Dependence, Sydney, Australia
Isselbacher, K J (1977) Metabolic and hepatic effects of alcohol New England Journal of Medicine *296* (11), 612—616
Jaarstatistiek van de in- en uitvoer per goederensoort van de Nederlandse Antillen (1962 through 1975) Bureau voor de Statistiek, Departement Sociale en Economisch Zaken, Curacao, Netherlands Antilles
Jackson, J K (1962) Alcoholism and the family In: D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley, London 616 + XVIII pp
Jansen, M M (1969) Alcohol en alcoholisme Stichting voor Alcohol Studies SOL Offsetdruk, Amsterdam, The Netherlands 53 pp
Janssen, J H G (1971) Some general aspects of alcoholism in Aruba Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 18—28 Verenigde Antilliaanse Drukkerijen, Aruba
Jellinek, E M (1942) "Death from alcoholism" in the United States in 1940 A statistical analysis Quarterly Jounal of Studies on Alcohol *3*, 465—494

Jellinck, E M (1945) Heredity of the alcoholic Quarterly Journal of Studies on Alcohol *6*, 105
Jellinek, E M (1951) In Expert Committee on Mental Health, WHO Report of the First Session of the Alcoholism Subcommittee, Annex 2 Jellinek Estimation Formula WHO Technical Reports Series no 42 24 pp
Jellinek, E M (1952) Phases of alcohol addiction Quarterly Journal of Studies on Alcohol *13* 673—684
Jellinek, E M (1959) Estimating the prevalence of alcoholism, modified values in the Jellinek formula and an alternative approach Quarterly Journal of Studies on Alcohol *20*, 261—269
Jellinek, F M (1960) The disease concept of alcoholism Hill House, New Haven 246 + X pp
Jellinek, F M (1962) Phases of alcohol addiction p 356-368 In D J Pittman & C R Snyder (1962) Society, Culture, and Drinking Patterns Wiley, London 616 + XVIII pp
Jones, H (1966) Alcoholisme Aulareeks Het Spectrum, Utrecht 216 pp
Jones, K L, Shainberg, L W & Byer, C O (1970) Drugs, Alcohol and Tobacco Canfield Press, San Francisco 113 + X pp
Jones, K I, Smith, D W & Hanson, J W (1976) The fetal alcohol syndrome Clinical delineation p 130—137 In F A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273* 130—137
Kalant, H & Kalant O J (1971) Drugs Society and Personal Choice PaperJacks, a division of General Publishing Co Ltd, Don Mills, Canada 160 + XVI pp
Kamphuisen, H A C, Jennekens F G I & Smalhout, B (1965) Formes dangereuses du délirium tremens Psychiatria, Neurologia, Neurochirurgia *68*, 360—370
Kaplan, H B, Pokorny, A D, Kanas, T & Lively, G (1974) Screening tests and self-identification in the detection of alcoholism Journal of Health & Social Behavior *15*, 51—56
Kellam, A M P & Wesolkowski, J (1968) A case of alcoholism treated by disulfiram implantation Journal of Alcoholism *3*, 38—40
Kellam, A M P (1969) Disulfiram implantation Journal of Alcoholism *4*, 228
Keller, M & Efron, V (1955) The prevalence of alcoholism Quarterly Journal of Studies on Alcohol *16*, 619—644
Keller, M (1962) The definition of alcoholism and the estimation of its prevalence p 310—329 In D J Pittman & C R Snyder (1962) Society, culture and drinking patterns Wiley, London 616 + XVIII pp
Keller, M (1972) On the loss of-control phenomenon in alcoholism British Journal on Addiction *67*, 153-166
Ketel, A P (1963) Gegevens betreffende alcoholzucht en ambulante behandeling van alcoholisten Ned Tijdschr v Geneesk *107*, 641—648
Klatsky, A L, Friedman, G D, Siegelaub, A B & Gerard, M J (1977) Alcohol consumption and blood pressure Kaiser Permanent Multiphasic Health Examination Data New England Journal of Medicine *296*, 1195—1200
Knupfer, G (1967) Epidemiologic studies and control programs in alcoholism V The epidemiology of problem drinking American Journal of Public Health *57*, 973—986
Knupfer, G & Room, R (1964) Age, sex and social class as factors in amount of drinking in a metropolitan community Social Problems *12* 224—240
Koch Weser, J (1974) Common poisons p 654—667 In M M Wintrobe et al (1974) Harrison's Principles of Internal Medicine McGraw-Hill Book Company, New York 2044 + XXIX + I 79 pp
Kraft, Th B (1974) Ervaringen met disulfiram implantaties, met beschrijving van een disulfirampsychose Tijdschrift voor Psychiatrie *16*, 126-132

Krimmel, H. E. (1973). The alcoholic and his family. p. 297—310. In: P. G. Bourne & R. Fox (1973): Alcoholism. Progress in Research and Treatment. Academic Press Inc., New York. 439 + XIV pp.
Laclé, C. (1971). The task of the social worker in the treatment of alcoholism. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p. 76-79. Verenigde Antilliaanse Drukkerijen, Aruba.
Latour, M. D. & Uittenboogaard, P. H. J. (1953). De taal Papiamento en haar oorsprong. Curaçao, Netherlands Antilles.
Lawrence, J. J. & Maxwell, M. A. (1962). Drinking and socio-economic status. p. 141—145. In: D. J. Pittman & C. R. Snyder (1962): Society, Culture and Drinking Patterns. Wiley, London. 616 + XVIII pp.
Leach, B. (1973). Does Alcoholics Anonymous really work? In: P. G. Bourne & R. Fox (1973): Alcoholism. Progress in Research and Treatment. Academic Press Inc., New York. 439 + XIV pp.
Ledermann, S. (1956). Alcohol, alcoolisme, alcoolisation; donnés scientifiques de caractère physiologique, économique et social. Institut National d' Etudes Démographiques. Travaux et Documents, Cahier No. 29. Presses Universitaires, Paris.
Lederman, S. (1964). Alcool, alcoolisme, alcoolisation; mortalité, morbidité, accidents du travail. Institut National d'Etudes Démographiques. Travaux et Documents, Cahier No. 41. Presses Universitaires, Paris.
Lelbach, W. K. (1966). Leberschaden bei chronischem Alkoholismus. Acta hep.-splenol. *13*, 321—349.
Lereboullet, J. (1964). Q'est-ce que l'alcoolisme? Revue du Practicien *14*, 359-374.
Lieber, C. S. (1973). Ethanol and the liver. In: P. G. Bourne & R. Fox (1973): Alcoholism. Progress in Research and Treatment. Academic Press Inc., New York. 439 + XIV pp.
Lieber, C. S. (1973a). Liver adaptation and injury in alcoholism. New England Journal of Medicine *228*, 356—362.
Lipscomb, W. R. & Sulka, M. (1961). Some factors affecting the geographic comparison of alcoholism prevalence rates; implications for the use of the Jellinek estimation formula. Quarterly Journal of Studies on Alcohol *22*, 588—596.
Liu, Y. K. (1973). Leukopenia in alcoholics. American Journal of Medicine *54*, 605—610.
Lolli, G. (1953). Alcoholism in women. Conn. Rev. Alcoholism *5*, 9—11.
Lundquist, G. A. R. (1972). Alkoholberoendets yttringar och förlopp. (Alcohol dependence - its symptoms and development) - Alkoholfragan *66*, 3-13.
Macdonald, D. E. (1956). Mental disorders in wives of alcoholics. Quarterly Journal of Studies on Alcohol *17*, 282—287.
Malcolm, M. T. (1972). The use of disulfiram implantation in the Liverpool region. 7 pp. Proceedings of the 30th International Congress on Alcoholism and Drug Dependence, Amsterdam.
Manual of statistic classification of diseases, injuries and causes of death; 7th revision (1955). WHO, Geneva.
Mardones, R. J. (1951). On the relationship between deficiency of B vitamins and alcohol intake in rats. Quarterly Journal of Studies on Alcohol *12*, 563—575.
Marie, C. (1955). A propos d'un nouveau mode de traitement de l'alcoolisme chronique par implantation de disulfure de tétra-éthyle-thio-urame. Thesis, Paris.
Mc. Clearn, G. E. (1973). The genetic aspects of alcoholism. p. 337-358. In: P. G. Bourne & R. Fox (1973): Alcoholism. Progress in Research and Treatment. Academic Press Inc., New York. 439 + XIV pp.
Mc Cord, W. & Mc Cord, J. (1962). A longitudinal study of the personality of alcoholics. p. 413—430. In: D. J. Pittman & C. R. Snyder (1962): Society, Culture and Drinking Patterns. Wiley, London. 616 + XVIII pp.

Mendelson, J. H. (1970). Biologic concomitants of alcoholism. (First of two parts.) New England J. of Med. *283*, 24—32.
Mendelson, J. H. (1970a). Biologic concomitants of alcoholism. (Second of two parts.) New England J. of Med. *283*, 71-81.
Mendelson, J., Wexler, D., Kutzansky, P., Leider, H. & Solomon, P. (1959). Serum magnesium in delirium tremens and alcoholic hallucinosis. J. Nerv. Ment. Dis. *128*, 352—357.
Merry, J. (1971). Alcohol (ethanol); some facts and figures. Practitioner *207*, 67—73.
Merry, J. (1971a). Cortisol response in alcoholics to ethanol, tranquillizers and sedatives. Proceedings of the 17th International Institute on the Prevention and Treatment of Alcoholism, West-Berlin.
Merry, J. & Marks, V. (1969). Plasma hydrocortisone response to ethanol in chronic alcoholics. Lancet *1*, 921—923.
Mertens, A. Th. L. M., Grosfeld, J. A. M., Morshuis, J. G. F., Persoon, J. M. G., Curfs, M. A. W., Dijkhuis-Braakhuis, G. B. J., Dijkman, A. J. T., van Gestel, W. J. C., Hammes, E. L. J., Heydendael, P. H. J. M., Kirkels, V. G. H. J., Michels, J. J. M., Neefs, W. J. M., van Thiel, T. A. M. G., Verschueren, M. J. M. (1965). De patiënt als gezinslid. Dekker & Van de Vegt, Utrecht-Nijmegen. 259 + VIII pp.
Miller, G. H. & Agnew, N. (1974). The Ledermann model of alcohol consumption. Description, implications and assessment. Quarterly Journal of Studies on Alcohol *35*, 877—898.
Milt, H. (1969). Basic Handbook on Alcoholism. Scientific Aids Publications, Maplewood, New Jersey, USA. 95 pp.
Mindlin, D. F. (1959). The characteristics of alcoholics as related to prediction of therapeutic outcome. Quarterly Journal of Studies on Alcohol *20*, 604-619.
Moore, R. A. (1971). The prevalence of alcoholism in a community general hospital. Amer. J. Psychiat. *128*, 638—639.
Moore, R. A. (1972). The diagnosis of alcoholism in a psychiatric hospital: a trial of the Michigan Alcoholism Screening Test (MAST). Amer. J. Psychiat. *128*, 1565—1569.
Mulford, H. A. (1964). Drinking and deviant drinking, U.S.A., 1963. Quarterly Journal of Studies on Alcohol *25*, 634—650.
Murray, R. M. (1976). Alcoholism amongst male doctors in Scotland. Lancet *2*, 729—731.
National Council on Alcoholism (1970). Facts on alcoholism. (leaflet).
Nordmo, S. M. (1959). Blood groups in schizophrenia, alcoholism and mental deficiency. Amer. J. Psychiat. *116*, 460.
Norris, J. L. (1976). Alcoholics Anonymous and other self-help groups. p. 735-776. In: R. E. Tarter & A. A. Sugerman (1976): Alcoholism. Interdisciplinary Approaches to an Enduring Problem. Addison-Wesley Publishing Company, Inc. Advanced Book Program. Reading, Massachusetts, U.S.A. 857 + XIV pp.
Oliemans, A. P. (1969). Morbiditeit in de huisartspraktijk. Stenfert Kroese, Leiden. 257 + XIV pp.
Oldenboom, J. F. (1971). Criminological and policial aspects of alcoholism in Aruba. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p. 96—99. Verenigde Antilliaanse Drukkerijen, Aruba.
Ouelette, E. M. & Rosett, H. L. (1976). A pilot prospective study of the fetal alcohol syndrome at the Boston City Hospital. Part II: the infants. p. 123-129. In: F. A. Seixas & S. Eggleston (1976): Work in Progress on Alcoholism. 664 + VIII pp. Annals of the New York Academy of Sciences *273*, 123-129.
Paige, P. E., La Pointe, W. & Krueger, A. (1971). The marital dyad as a diagnostic and treatment variable in alcohol addiction. Psychology (Savannah) *8*, 64—73.
Paillot, M. & Jacques, D. (1968). Approche thérapeutique de l'alcoolisme chronique par la méthode d'un implant sous-cutané de Disulfirame. Rev. Hyg. et Méd. soc. *16*, 689—698.

Palola, E G, Dorpat, Th I & Larson, W R (1962) Alcoholism and suicidal behavior p 511—534 In D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley, London 616 + XVIII pp
Partanen, J, Bruun, K & Markkanen T (1966) Inheritance of Drinking Behavior. A Study on Intelligence, Personality and Use of Alcohol of Adult Twins Alcohol Research in the Northern Countries, Vol 14 The Finnish Foundation for Alcohol Studies, Helsinki 159 pp
Parzen, E (1960) Modern probability theory and its applications Wiley, London
Paunier L, Radde, I C, Kolh, S W, Conen, P F & Fraser, D (1968) Primary hypomagnesaemia with secondary hypocalcaemia in an infant Pediatrics *41*, 385—402
Pawan, G L S (1970) Sucrose and fructose increase alcohol metabolism Physician's Alcohol Newsletter *5*, no ?
Péquignot, G (1958) Enquête par interrogatoire sur les circonstances diététiques de la cirrhose alcoolique en France Bull Inst Nat Hyg *13*, 719—739
Perman, F S (1962) Studies on the antabuse alcohol reaction in rabbits Acta physiologica scandinavica *55*, suppl 190, 1—46
Pijnenburg, Th P (1971) Identification problems in alcoholics and other addicts Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 106—110 Verenigde Antilliaanse Drukkerijen, Aruba
Pinto, H (1963) Group therapy among alcoholics p 169—176 In. Proceedings of the 4th Caribbean Conference for Mental Health, Curaçao Boek- en Offset Drukkerij "De Curaçaosche Courant", Curaçao 265 pp
Pittman, D J & Gordon, C W (1962) Criminal careers of the chronic drunkenness offender p 535—546 In D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley, London 616 + XVIII pp.
Plaut, Th F A (1967) Alcohol problems A report to the nation by the Cooperative Commission on the Study of Alcoholism Oxford University Press, New York 200 + XVI pp
Pokorny, A D, Miller, B A, Kaplan, H B (1972) The brief MAST a shortened version of the Michigan Alcoholism Screening Test Amer J Psychiat *129*, 342—345
Polco Conde, M (1971) Aspectos del tratamiento del alcoholismo en Venezuela. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 56-69 Verenigde Antilliaanse Drukkerijen, Aruba
Popham, R E (1956) The Jellinek alcoholism estimation formula and its application to Canadian data Quarterly Journal of Studies on Alcohol *17*, 559—593
Prigent, M L (1960) Place de l'application du disulfure de tétra-éthyle-thiourame en implants dans le traitement de l'alcoolisme chronique Thesis, Rennes
Produktschap voor Gedistilleerde Dranken (1971) Hoeveel alcoholhoudende dranken worden er in de wereld gedronken ? Produktschap voor Gedistilleerde Dranken, Schiedam 63 pp
Produktschap voor Gedistilleerde Dranken (1975) Hoeveel alcoholhoudende dranken worden er in de wereld gedronken ? Produktschap voor Gedistilleerde Dranken, Schiedam 63 pp
Produktschap voor Gedistilleerde Dranken (1976) Hoeveel alcoholhoudende dranken worden er in de wereld gedronken ? Produktschap voor Gedistilleerde Dranken, Schiedam 63 pp
Pytkowicz Streissguth, A (1976) Psychologic handicaps in children with the fetal alcohol syndrome p 140—145 In F A Seixas & S Eggleston (1976)· Work in Progress on Alcoholism 644 + VIII pp Annals of the New York Academy of Sciences *273*, 140—145
Que, G S (1975) Beschadiging van organen door alcoholmisbruik T Alc Drugs *1*, 41—46
Rankin, J G (1969) Definitive treatment of alcoholism p 31—39 In Symposium Alcoholism - problems in treatment University of Melbourne, Australia

Rathod, N H, Gregory, E, Blows, D & Thomas, G H (1966) British Journal of Psychiatry *112*, 683
Rieth, E (1970) Die soziologischen und psychologischen Ursachen der Sucht bei Frauen p 34—41 In R Battegay et al (1970) Alkoholismus bei Frauen Hoheneck Verlag GmbH, Hamm, Germany 96 pp
Robins, L N, Bates, W M & O'Neal, P (1962) Adult drinking patterns of former problem children p 395—411 In D J Pittman & C R Snyder (1962) Society, Culture and Drinking Patterns Wiley, London 616 + XVIII pp
Roe, A (1944) The adult adjustment of children of alcoholic parents raised in foster homes Quarterly Journal of Studies on Alcohol *5*, 378-393
Roos, J (1976) Alcohol consumptie als oorzaak van ziekten buiten de lever. Huisarts en Wetenschap *19*, 21—23
Rosett, H L (1976) Effects of maternal drinking on child development: an introductory review p 115—117 In I A Seixas & S Eggleston (1976). Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 115—117
Rosett, H L, Ouclette, E M & Weiner, L (1976) A pilot prospective study of the fetal alcohol syndrome at the Boston City Hospital Part I maternal drinking p 118—122 In I A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 118—122
Salel, A F, Tong, A, Zelis, R, Miller, R R, Borhani, N O & Mason, D T, (1977) Accuracy of numerical coronary profile correlation of risk factors with arterio-graphically documented severity of atherosclerosis New England Journal of Medicine *296*, 1447—1450
Santamaria, J N (1969) Treatment of the 'incurable' alcoholic patient p. 44—49. In Symposium Alcoholism problems in treatment University of Melbourne, Australia
Sassoon, H F & White, J B (1970) Alcoholism and colour vision are there familial links? Lancet, 367—368
Savage, D C L & Mc Adam, W A F (1967) Convulsions due to hypomagnesaemia in an infant recovering from diarrhoea Lancet *2*, 234—236
Sayres, W C (1956) Ritual drinking, ethnic status and inebriety in rural Colombia Quarterly Journal of Studies on Alcohol *17*, 53—62
Schmidt, W & De Lint, J (1970) Estimating the prevalence of alcoholism from alcohol consumption and mortality data Quarterly Journal of Studies on Alcohol *31*, 957—964
Schmidt, W & De Lint, J (1972) Causes of death of alcoholics Quarterly Journal of Studies on Alcohol *33*, 171—186
Schmidt, W & Popham, R E (1968) Alcohol consumption of alcoholics Addiction Research Foundation, Toronto, Canada
Schouten, J A M (1963) Deficient father identification and the development of delinquent behaviour p 71—87 In Proceedings of the 4th Caribbean Conference for Mental Health, Curaçao Boek en Offset Drukkerij "De Curaçaosche Courant", Curaçao 265 pp
Seevers, M H (1962) Medical perspectives on habituation and addiction JAMA *181*, 92—98
Selye, H (1946) General adaptation syndrome and diseases of adaptation J Clin Endocrin *6*, 117—230
Selzer, M L (1971) The Michigan Alcoholism Screening Test, the quest for a new diagnostic instrument Amer J Psychiat *127*, 1653—1658
Selzer, M L, Vinokur, A & Van Rooijen, M A (1975) A self-administered short Michigan Alcoholism Screening Test (SMAST) Journal of Studies on Alcohol *36*, 117—126
Sijlbing, G (1977) Craving, loss of-control en matig drinken Therapeutische konsekwenties voor de behandeling van alkoholisten T Alc Drugs *3*, 15—20.

Skog, O J (1973) Less alcohol - fewer alcoholics ? Drinking & Drug Pract Surveyor, Berkeley, No 7, 7—14
Sluyter, D P, Arntzenius, A C, Meijer, J & Styblo, K (1977) CB Heart Project in The Netherlands Population under study and methods of intervention Hart Bulletin *8*, 42—46
Smart, R D (1965) Effects of alcohol on conflict and avoidance behavior Quarterly Journal of Studies on Alcohol *26*, 187—205
Smith, D W, Jones, K L & Hanson, J W (1976) Perspectives on the cause and frequency of the fetal alcolic syndrome p 138—139 In F A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 138—139
Smith, J F B & Lucie, N P (1973) Alcohol - a cause of stress erythrocytosis ? Lancet *1*, 637 638
Smith, J J (1949) A medical approach to problem drinking Quarterly Journal of Studies on Alcohol *10*, 251—257
Solms, H (1976) Alcoholism in Europe In F A Seixas & S Eggleston (1976) Work in Progress on Alcoholism 664 + VIII pp Annals of the New York Academy of Sciences *273*, 24—32
Spodick, D H, Pigott, V M & Chirife, R (1972) Preclinical cardiac malfunction in chronic alcoholism New England Journal of Medicine *287*, 677—680
Statistisch Jaarboek Nederlandse Antillen (1971) Bureau voor de Statistiek, Departement Sociale en Economische Zaken, Curaçao, Netherlands Antilles
Statistisch Jaarboek Nederlandse Antillen (1974) Bureau voor de Statistiek, Department Sociale en Economische Zaken, Curaçao, Netherlands Antilles
Statistische Mededelingen Nederlandse Antillen (1970 through 1976) Bureau voor de Statistiek, Departement Sociale en Economische Zaken, Curaçao, Netherlands Antilles
Steenmeyer, F (1957) Food and nutrition of Arubans Thesis, Utrecht 139 + VIII pp
Sturmans, F & Mulder, P G H (1976) De bruikbaarheid van een methode voor epidemiologisch onderzoek Tijdschrift voor Sociale Geneeskunde *54*, 404—408
Sturmans, F & Mulder, P G H (1976a) Enkele fundamentele frequentiematen in de epidemiologie Tijdschrift voor Sociale Geneeskunde *54*, 277—281
Sturmans, F & Mulder, P G H (1976b) De betekenis van de prevalentie voor de predictieve waarde van een test Tijdschrift voor Sociale Geneeskunde *54*, 496—501
Styblo, K, Meijer, J, Arntzenius, A C, Haas, J H de, Geuns, H A van, Mellema, T L & Sluyter, D P (1977) C B Heart Project in The Netherlands Results of intervention in high risk individuals Hart Bulletin *8*, 47 59
Thuline, H C (1967) Inheritance of alcoholism Lancet *1*, 274
Tinklenberg, J R (1973) Alcohol and violence p 195—210 In P G Bourne en R Fox (1973) Alcoholism Progress in Research and Treatment Academic Press Inc, New York 439 + XIV pp
Tintera, J W & Lovell, H W (1949) Endocrine treatment of alcoholism Geriatrics *4*, 274—280
Truitt, E B & Duritz, G (1967) The role of acetaldehyde in the actions of ethanol In R P Maickel (1967) Biochemical Factors in Alcoholism Pergamon Press, Oxford 256 + XI pp
Turfboer, R (1957) The effects of in-plant rehabilitation of alcoholics Med Bulletin, 108—128
Turfboer, R (1971) The typical Aruban alcoholic in the nineteen fifties. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p 70 75 Verenigde Antilliaanse Drukkerijen, Aruba
Van Epen, J H (1974) Compendium Drugverslaving en Alcoholisme Diagnostiek en behandeling Agon Elsevier, Amsterdam/Brussels 188 + XII pp

Van Erp, Th. (1971). Possibilities of the implant of disulfiram (Esperal). Proceedings of the 17th International Institute on the Prevention and Treatment of Alcoholism, West-Berlin.
Van Zonneveld, R. J. et al. (1972). Sociale Geneeskunde. Een algemene inleiding. Academische Paperbacks. A. Oosthoek's Uitgeversmaatschappij N.V., Utrecht. 486 + XII pp.
Van de Voort, P. P. C. H. (1971). Some juridical considerations in defence of the Aruban alcoholic. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p. 188—191. Verenigde Antilliaanse Drukkerijen, Aruba.
Van der Does de Willebois, A. E. M. (1965). Vervreemding en verslaving. Dekker & van de Vegt, Utrecht. 229 + VIII pp.
Victor, M. (1973). The role of hypomagnesemia and respiratory alkalosis in the genesis of alcohol-withdrawal symptoms. p. 235—248. In: F. A. Seixas & S. Eggleston (1973): Alcoholism and the Central Nervous System. 389 pp. Annals of the New York Academy of Sciences *215*, 235—248.
Victor, M. & Adams, R. D. (1974). Alcohol. p. 671—681. In: M. M. Wintrobe et al. (1974): Harrison's Principles of Internal Medicine. Mc Graw-Hill Book Company, New York. 2044 + XXIX + I 79 pp.
Victor, M. & Wolfe, S. M. (1973) Causation and treatment of the alcohol withdrawal syndrome. p. 137—169. In: P. G. Bourne & R. Fox: Alcoholism. Progress in Research and Treatment (1973). Academic Press Inc., New York. 439 + XIV pp.
Vismans, R. (1971). Pastoral care and alcoholism in Aruba. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p. 80—87. Verenigde Antilliaanse Drukkerijen, Aruba.
Vogel-Sprott, M. (1974). Defining "light" and "heavy" social drinking; research implications and hypotheses. Quarterly Journal of Studies on Alcohol *35*, 1388—1392.
Wahl, C. W. (1956). Some antecedent factors in the family histories of 109 alcoholics. Quarterly Journal of Studies on Alcohol *17*, 643—654.
Wallace, J. G. (1972). Drinkers and abstainers in Norway. A national survey. Quarterly Journal of Studies on Alcohol, Supplement No. 6, 129—151.
Wallerstein, R. S. (1968). Alcoholism: symptom or disease? Int. J. Psychiat. *5*, 59—65.
Walsh, M. J. (1973). The biochemical aspects of alcoholism. In: P. G. Bourne & R. Fox (1973): Alcoholism. Progress in Research and Treatment. Academic Press Inc., New York. 439 + XIV pp.
Wanberg, K. W. (1969). Prevalence of symptoms found among excessive drinkers. International Journal of the Addictions *4*, 169—185.
Wanberg, K. W. & Knapp, J. (1970). A multidimensional model for the research and treatment of alcoholism. International Journal of the Addictions *5*, 69-97.
Wever, O. R. (1971)., Some medical and sociopathological aspects of alcoholism in Aruba and its actual treatment. Proceedings of the First Aruban & Antillean Congress on Alcoholism, p. 135—187. Verenigde Antilliaanse Drukkerijen, Aruba.
Wever, O. R. (1971a). Brief report on alcoholism in Aruba. Proceedings of the 17th International Institute on the Prevention and Treatment of Alcoholism, West-Berlin. 11 pp.
Wever, O. R. (1975). Over het alcoholisme op Aruba. Nieuwe West-Indische Gids *50*, 89—106.
Wever, O. R. (1976). Methoden ter schatting van het vóórkomen van alcoholisme in een bevolking. T. Alc. Drugs *2*, 19—25.
Wever, O. R. (1976a). Jaarlijkse alcoholconsumptie, sterfte tengevolge van alcoholische levercirrose en het vóórkomen van alcoholisme op Aruba, 1961—1973. T. Alc. Drugs *2*, 57—66.
Wever, O. R. & Gips, C. H. (1977). Schatting van het aantal excessieve drinkers en alcoholisten in Nederland met de Ledermann-formule, 1875 tot 1977. T. Alc. Drugs *3*, 37—43.

Wexberg, I E (1951) Alcoholism as a sickness Quarterly Journal of Studies on Alcohol *12*, 217—230
Whalen, Th (1953) Wives of alcoholics four types observed in a family service agency Quarterly Journal of Studies on Alcohol *14*, 632—641
Wilkins, R H (1973) Alcoholism clues to the diagnosis in general practice Update *6*, 1797—1804
Wilkins, R H (1975) The hidden alcoholic in general practice British Journal of Addiction *70*, Suppl Conference Alcoholism, 12—17
Wilkins, R H (1975a) An alcoholic patient in general practice - detection and subsequent management 4 pp Paper published by Medical Council on Alcoholism, London
Wilkins, R H (1976) Waarom moeite doen om potentiele alkoholisten op te sporen ? T Alc Drugs *2*, 134 –136
Wilkins, R H (1976a) Alcoholism the GP's role Medicine *13*, 593—594
Wilkins, R H (1977) An alcoholic at risk register in general practice - why bother ? Der Praktische Arzt *4*, 661—664
Wilkinson, P , Santamaria, J N , Rankin, J G & Martin, D (1969) Epidemiology of alcoholism, social data and drinking patterns of a sample of Australian alcoholics Med J Australia *1*, 1020—1025
Williams, R H (1974) Textbook of Endocrinology 5th Edition W B Saunders Company, Philadelphia 1138 + XXIV pp
Williams, R J (1947) The etiology of alcoholism, a working hypothesis involving the interplay of hereditary and environmental factors Quarterly Journal of Studies on Alcohol *7*, 567—587
Zung, B J & Charalampous, K D (1975) Item analysis of the Michigan Alcoholism Screening Test Journal of Studies on Alcohol *36*, 127—132

APPENDIX A

QUESTIONNAIRE DRINKING AND SMOKING HABITS

Foundation against Alcohol-abuse Aruba

Name of interviewed person:

Number of interviewed person:

---

* A) Number of interviewed person:
* B) Date:
* C) Name of interviewer:
* D) Address:
* E) Date of birth:
* F) Birthplace:
G) Date of birth of marital partner:
H) Total number of persons in family or family-unit:
I) How many living children do you have?
Of this marriage:
Of earlier marriage(s):
Out of wedlock:
J) What is the occupation of the head of the family?
K) What is or was your father's occupation?
L) What is your occupation?
M) What kind of alcohol-containing drinks do you take and how much on average on the following days?
The estimated average as given by the interviewed person has to be noted every time.

Friday		*Saturday*		*Sunday*
Monday	*Tuesday*	*Wednesday*	*Thursday*	*TOTAL:*

Daily average on religious and national holidays (2 Christmas days, New Year's day, 3 Carnival days, 3 days Easter, the Queen's birthday, Ascension day, 3 days Pentecost):

TOTAL:
(= 14 x average)

Average daily consumption on a weekday: (= A)
Average daily consumption on a holiday: (= B)
365 x A = (1)
14 x (B—A) = (2)

(1) + (2) = (3) = *Annual consumption*

N.B. If interviewed person usually drinks together with several others, his share has to be calculated by dividing the total amount by the average number of persons that partake in consuming this amount.

	Variable No.	*Score*
*	1) District (see sub D) by constituency (voting-district):	
	(1) Oranjestad	1
	(2) Nort	2
	(3) Paradera	2
	(4) Santa Cruz	2
	(5) Sabaneta & Brazil	2
	(6) San Nicolás	1

* 2 } Age:
* 3 }

(0)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
(0)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)

* 4) Nationality (as given by the interviewed person on the question "What is your nationality?"):

(1) Aruban	1
(2) Bonairean	0
(3) Curaçaoan	0

(4)	Windward Islander	0
(5)	Surinamer	0
(6)	(European-) Dutch	0
(7)	Venezuelan	0
(8)	Latin-American	0
(9)	American (U.S.A.)	0
(10)	Other	0
(11)	Not applicable	0

* 5) Sex:

(1)	Male	2
(2)	Female	1

6) Civil status:

(1)	Married	1
(2)	Common-law	2
(3)	Divorced (not living in common-law)	3
(4)	Widower/widow, not living in common-law	3
(5)	Unmarried	3
(6)	Not applicable	0

7) Religion:

(1)	Roman Catholic	1
(2)	Protestant	1
(3)	Jewish	1
(4)	Other	1
(5)	No religion	0
(6)	Not applicable	1 (= mode)

8) Type of dwelling:

(1)	Brick house	1
(2)	Wooden or corrugated iron house	2
(3)	Mud cabin	3
(4)	Slum dwelling	3
(5)	Not applicable	0

9) Who is head of the family?

(1) You	0
(2) Your marital partner	1
(3) Your father	0
(4) Your mother	0
(5) Someone else	1
(6) Not applicable	0

10) Are you at present:

(1) Unemployed	3
(2) Student	2
(3) Housewife	2
(4) Pensioner	2
(5) Irregularly employed	2
(6) Regularly employed	1
(7) Not applicable	1 (= mode)

11) Do you have at present:

(1) No income	5
(2) Unemployment pay	4
(3) Insufficient income for a decent living	3
(4) Sufficient income for a decent living	2
(5) More than sufficient income for a decent living	1
(6) Not applicable	3 (= mode)

12) Amount of the total monthly income of the family:

(1) NAf. 0-100	5
(2) NAf. 100-200	4
(3) NAf. 200-500	3
(4) NAf. 500-1000	2
(5) NAf. 1000 or more	1
(6) Not applicable	3 (= mode)

13) How many hours a day do you work on an average?

(1) 0 hours	0
(2) 1-4 hours	1
(3) 4-8 hours	2
(4) More than 8 hours	3
(5) Not applicable	2 (= mode)

14) Do you enjoy your work?

(1) Yes	1	
(2) No	3	
(3) Don't know	2	
(4) Not applicable	1	(= mode)

15) Do you have a lot to do at work?

(1) Yes	3	
(2) No	1	
(3) Don't know	2	
(4) Not applicable	1	(= mode)

16) Are you satisfied with the position you've reached in life?

(1) Yes	1	
(2) Fairly	2	
(3) No	3	
(4) Not applicable	1	(= mode)

17) Are you satisfied with your health?

(1) Yes	1	
(2) Fairly	2	
(3) No	3	
(4) Not applicable	1	(= mode)

18) Do you consider the living space in your house:

(1) Adequate	1	
(2) Barely adequate	2	
(3) Inadequate	3	
(4) Not applicable	1	(= mode)

19) How much does your family approximately spend every month on beverage alcohol?

(1) NAf. 0	1	
(2) NAf. 0-50	2	
(3) NAf. 50-100	3	
(4) NAf. 100 or more	4	
(5) Don't know/Not applicable	0	

20) What schooling have you had?

(1)	No schooling at all	6
(2)	A few years elementary school	5
(3)	Elementary school completed	4
(4)	Technical school or school of domestic science	3
(5)	Higher-grade school or Polytechnic school	2
(6)	Secondary grammar school, teacher training academy	1
(7)	College of social studies, art academy, College of Advanced Technology, College training	1
(8)	University	1
(9)	Not applicable	4 (= mode)

* 21) How many cups of coffee or tea do you drink daily?

(1)	None	0
(2)	1-2	1
(3)	2-5	2
(4)	5 or more	3
(5)	Not applicable	1 (= mode)

* 22) How often do you eat biscuits, pies or other sweets?

(1)	Never	0
(2)	Seldom	1
(3)	On holidays	2
(4)	Every second day	3
(5)	Daily	4
(6)	Not applicable	1 (= mode)

* 23) Are there certain times at which you tend to eat (more) sweets?

(1)	Never	0
(2)	Seldom	1

(3) On holidays 2
(4) During week-ends 3
(5) At work 4
(6) Before, during or after meals 4
(7) After work 4
(8) Not applicable 0

* 24) What do you usually smoke?
(1) Cigarettes 1
(2) Cigars 1
(3) A pipe 1
(4) Nothing 0
(5) Not applicable 0

* 25) How much do you usually smoke a day?
(1) 1- 5 cigarettes 1
(2) 5-10 cigarettes 2
(3) 10-20 cigarettes 3
(4) More than 20 cigarettes 4
(5) 1- 5 cigars 1
(6) 5-10 cigars 2
(7) More than 10 cigars 3
(8) Nothing, not applicable 0

26) What induces you to smoke more than usual?
(1) Nothing 0
(2) Having a good time with people 1
(3) Tension at home or at work 1
(4) Illness 1
(5) Inner fears, anxiety 1
(6) Boredom 1
(7) Uncertainty, lack of self-confidence 1
(8) Restlessness 1
(9) Depression 1
(10) Not applicable 0

* 27) On which day of the week do you usually smoke most?

(1) Friday	2
(2) Saturday	2
(3) Sunday	2
(4) Monday	1
(5) Tuesday	1
(6) Wednesday	1
(7) Thursday	1
(8) Not applicable	0

28) At what age did you start smoking?

(1) 10-15 years
(2) 15-20 years
(3) 20-25 years
(4) 25 years or older
(5) Not applicable

29) Did you have permission of your parents or those taking care of you when you first started smoking?

(1) Yes	1
(2) No	2
(3) Not applicable	0

* 30) What do you usually choose when you are offered a drink?

(1) Wine, Sherry or Vermouth	2
(2) Beer	1
(3) Rhum	3
(4) Brandy	3
(5) Cucuy**	3
(6) Whisky	3
(7) Liqueur	3
(8) Gin or other hard liquor	3
(9) Cola	0

** *Cucuy is a typically Aruban liqueur.*

(10) Juice or other softdrinks 0
(11) Not applicable 0

* 31) Do you ever offer a drink to someone?
(1) Regularly 3
(2) Occasionally 2
(3) Seldom 1
(4) Never 0
(5) Not applicable 0

32) If so, where?
(1) At home 0
(2) Bar, canteen, restaurant 2
(3) Out of the house (parranda) 2
(4) Not applicable 0

33) Do you usually drink:
(1) Alone 2
(2) With members of the family 0
(3) With relatives 0
(4) With friends 2
(5) With colleagues from work 2
(6) With "drinking-friends" 3
(7) Not applicable 0

* 34) On which day of the week do you usually drink most?
(1) Friday 2
(2) Saturday 2
(3) Sunday 2
(4) Monday 1
(5) Tuesday 1
(6) Wednesday 1
(7) Thursday 1
(8) Not applicable 0

35) If you never take any alcohol-containing beverages, why don't you?

(1) No financial means	1
(2) Dislike of alcohol	1
(3) Alcohol makes me sick	1
(4) Religious reasons	1
(5) Fear of becoming an alcoholic	1
(6) No taste for alcohol	1
(7) Other motives	1
(8) Not applicable	0

* 36) How often do you drink alcohol-containing beverages?

(1) Never	0
(2) Once a year or less	1
(3) At parties only	3
(4) Once every two months	2
(5) At most once a week	4
(6) Every week-end	5
(7) 2-3 times a week	5
(8) Every day	6
(9) Not applicable	0

* 37) How often are you drunk? (I.e. before you were ever treated for alcoholism.)

(1) Never	0
(2) Once a year or less	1
(3) At parties only	3
(4) Once every two months	2
(5) At most once a week	4
(6) Every week-end	5
(7) 2-3 times a week	5
(8) Every day	6
(9) Not applicable	0

38) At what age did you start to drink alcohol-containing beverages regularly - i.e. apart from the occasional sip when you were a child?

(1) 10-15 years
(2) 15-20 years
(3) 20-25 years
(4) 25 years or older
(5) Not applicable

39) At what age were you drunk for the first time?
(1) 10-15 years
(2) 15-20 years
(3) 20-25 years
(4) 25-30 years
(5) 30-35 years
(6) 35 years or older
(7) Not applicable

40) For how many years were you a so-called "social drinker" before your alcohol problem became manifest?
(1) 0- 1 years
(2) 1- 3 years
(3) 3- 5 years
(4) 5-10 years
(5) 10-15 years
(6) 15-20 years
(7) More than 20 years
(8) Not applicable

41) Do you drink a lot in connection with your work or with people you know from work?
(1) Regularly 3
(2) Occasionally 2
(3) Never 1
(4) Not applicable 0

42) Do you ever take a drink too many?
(1) Regularly 3
(2) Occasionally 2
(3) Never 1
(4) Not applicable 0

43) If so, how do you usually feel when this happens?

(1) Cheerful	1
(2) Relaxed	1
(3) A real man	1
(4) Without fears	1
(5) Abandoned	1
(6) Sick	1
(7) Depressed	1
(8) Different (not mentioned here)	1
(9) Not applicable	0

44) Did you drink more, or less in the past than now, or as much?

(1) More	2
(2) The same amount	1
(3) Less	2
(4) Not applicable	0

45) Why do you actually drink alcoholic beverages?

(1) Because others drink, and I don't want to be the odd one out	1
(2) Because I like drinking	1
(3) Because I think it healthy	1
(4) Because I consider it a real masculine habit	1
(5) Because I feel better when I am drinking	1
(6) Because it gives me more self-confidence	1
(7) To forget troubles and misery	1
(8) No reasons/other reasons	0
(9) Not applicable	0

* 46) What would you think if someone of your family would be drunk occasionally?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

* 47) What would you think if a member of your family would be "tipsy" occasionally?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

* 48) What would you think if someone of your family would be drunk every week?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

49) What would you think if someone of your family would have a drink with the daily meals?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

* 50) What would you think if someone of your family would be drinking regularly without ever getting really drunk?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

* 51) What would you think if someone of your family would be getting drunk every night?

(1) Objectionable	3
(2) Doesn't interest me (very much)	2
(3) Normal	1
(4) Not applicable	3 (= mode)

* 52) What would you think if someone of your family on principe never would drink?

(1) Objectionable	1
(2) Doesn't interest me (very much)	2
(3) Normal	3
(4) Not applicable	3 (= mode)

53) Do you take (excessively) many alcohol-containing drinks on the following occasions, i.e. more than on ordinary days? (Cultural crisis of alcohol consumption.)
On national holidays, like the Queen's birthday, for example?

(1) Yes	1
(2) No	0
(3) Not applicable	0

54) On religious feast-days, like Christmas, Easter, Pentecost, for example?

(1) Yes	1
(2) No	0
(3) Not applicable	0

55) On family-celebrations?

(1) Yes	1
(2) No	0
(3) Not applicable	0

56) On pay-days?

(1) Yes	1
(2) No	0
(3) Not applicable	0

57) During the carnival?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 58) Do you drink (excessively) large amounts of alcohol-containing beverages because of inner fear, tiredness, tension, depression, boredom, uncertainty, lack of self-confidence, illness? (Pathological crisis of alcohol consumption; psychological dependence.)

(1) Yes	1
(2) No	0
(3) Not applicable	0

59) At what age did you start using alcohol in this manner?

(1) 15-20 years
(2) 20-25 years
(3) 25-30 years
(4) 30-35 years
(5) 35-40 years
(6) 40-45 years
(7) 45-50 years
(8) 50 years or older
(9) Not applicable

60) If so, how often do you drink for the above-mentioned reasons?

(1) Never	0
(2) Once a year or less	1
(3) 1-6 times a year	2
(4) At most once a month	3
(5) Weekly	4
(6) Daily	5
(7) Not applicable	0

61) How long do your drinking bouts last on average?

(1) 1-2 days	1
(2) 3-7 days	2
(3) 3-7 weeks	3
(4) 3-7 months	4
(5) More than a year	5
(6) Not applicable	0

* 62) Has it ever happened to you, when you started drinking again - after a shorter or longer period "on the wagon" - that you conldn't stop drinking after the first or the first few drinks, and that you had to continue drinking until you were completely drunk, sick, or had to vomit? ("Loss-of-control.")

(1) Never	0
(2) Regularly	1
(3) Always	1
(4) Not applicable	0

63) At what age did you first notice this?

(1) 15-20 years
(2) 20-25 years
(3) 25-30 years
(4) 30-35 years
(5) 35-40 years
(6) 40-45 years
(7) 45-50 years
(8) 50 years or older
(9) Not applicable

* 64) Do you have any of the following so-called withdrawal symptoms (discomfort, irritability, restlessness, tremulousness, palpitations, nausea, vomiting, anxiety, insomnia, hearing voices, seeing "ghosts") after having stopped drinking? (Physical dependence.)

(1) Never	0
(2) Probably	1
(3) Occasionally	1
(4) Regularly, not seriously (discomfort, irritability, restlessness)	1
(5) Regularly, seriously (tremulousness, palpitations, nausea, vomiting, anxiety, insomnia, seeing "ghosts", hearing voices)	2
(6) Not applicable	0

* 65) Do you drink before breakfast?

(1) Never 0
(2) Occasionally 1
(3) Regularly 1
(4) Not applicable 0

* 66) If so, why do you take a drink before breakfast?

(1) From sheer force of habit 0
(2) To get rid of a "hangover" 1
(3) Not applicable 0

* 67) Has it ever happened to you after you had been drinking, that you couldn't remember anything about the night before? ("Blackout".)

(1) Never 0
(2) Occasionally 1
(3) Regularly 1
(4) Always 1
(5) Not applicable 0

68) At what age did this first occur?

(1) 15-20 years
(2) 20-25 years
(3) 25-30 years
(4) 30-35 years
(5) 35-40 years
(6) 40-45 years
(7) 45-50 years
(8) 50 years or older
(9) Not applicable

* 69) Has your drinking ever been accompanied by an upset stomach or an inflammation of the stomach? (Gastritis alcoholica.)

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 70) Has your drinking ever been accompanied by inflammations of nerves in arm or leg? (Polyneuritis alcoholica.)

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 71) Has you drinking ever been accompanied by the hearing of non-existing voices? (Hallucinosis alcoholica.)

(1) Yes 2
(2) No 0
(3) Not applicable 0

* 72) Has you drinking ever been accompanied by delirium tremens? (D.T.'s, i.e., hearing of voices or seeing of things which do not exist, anxiety, excitation, and tremulousness.)

(1) Yes 2
(2) No 0
(3) Not applicable 0

* 73) Has your drinking ever been accompanied by convulsions or "fits"? (Epilepsia alcoholica.)

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 74) Has your drinking ever been accompanied by insanity, madness?

(1) Yes 1
(2) No 0
(3) Not applicable 0

75) Have you ever been treated for alcoholism or for diseases caused by alcohol?

(1) No 0
(2) Yes, just withholding alcohol ("detoxification") 1

(3) Yes, Refusal or Antabuse-tablets 1
(4) Yes, cure with test-drinks (Refusal-tablets or injections with apomorphine or emetine, followed by test-drinks) 1
(5) Yes, only group therapy 1
(6) Yes, only Alcoholics Anonymous 1
(7) Not applicable 0

76) If so, where were you treated at the time?
(1) San Pedro Hospital 1
(2) Lago Hospital 1
(3) At home, ambulatory patient 1
(4) At home, confined to bed 1
(5) Not applicable 0

77) Do you often have fears?
(1) Yes 1
(2) No 0
(3) Not applicable 0

78) If so, what do you fear most?
(1) Illness of yourself or of members of your family 1
(2) Poverty in your family 1
(3) Marriage- and family problems 1
(4) The future of your family 1
(5) Death 1
(6) To become or to be an alcoholic 1
(7) The pressure of life 1
(8) The possible loss of your job 1
(9) Not applicable 0

79) Do you often think about your religion?
(1) Yes 1
(2) No 0
(3) Not applicable 1 (= mode)

80) How often do you go to church?

(1) Never	0
(2) Seldom	1
(3) Only on religious holidays	2
(4) Every Sunday, or sabbath	3
(5) Also several times a week	4
(6) Not applicable	1 (= mode)

81) Do you ever talk with somebody about your religion?

(1) Yes	1
(2) No	0
(3) Not applicable	1 (= mode)

82) If so, with whom mostly?

(1) (Marital) partner	1
(2) Children	1
(3) Relatives	1
(4) Friends and acquaintances	1
(5) Co-religionists	1
(6) Not applicable	0

83) Do you feel sufficiently appreciated by the people in your surroundings?

(1) Yes	1
(2) Fairly	2
(3) No	3
(4) Not applicable	0

84) Have there been people in your life whom you appreciated very much and who let you down?

(1) Yes	1
(2) No	0
(3) Not applicable	0

85) If so, was this your (marital) partner or your fiancé(e)?

(1) Yes 1
(2) No 0
(3) Not applicable 0

86) If so, was this your father?
(1) Yes 1
(2) No 0
(3) Not applicable 0

87) If so, was this your mother?
(1) Yes 1
(2) No 0
(3) Not applicable 0

88) If so, were these your children?
(1) Yes 1
(2) No 0
(3) Not applicable 0

89) If so, were these other relatives?
(1) Yes 1
(2) No 0
(3) Not applicable 0

90) If so, were these friends or acquaintances?
(1) Yes 1
(2) No 0
(3) Not applicable 0

91) In what have they disappointed you most?
(1) Betrayal 1
(2) (Marital) infidelity 1
(3) Gossip, slander or libel 1
(4) Financial injury 1
(5) Not applicable 0

92) When you became aware of this, what did you do:

(1) Cry	1
(2) Sleep badly	1
(3) Get headaches or other physical complaints	1
(4) Seek oblivion in drugs	1
(5) Seek oblivion in alcohol	1
(6) Seek comfort with parents	1
(7) Smoke more	1
(8) Seek comfort with friends	1
(9) Not applicable	0

93) When you're thinking of your youth, when you were still living with your parents, do you have:

(1) Pleasant memories	1
(2) Pleasant and unpleasant memories	2
(3) Only unpleasant memories	3
(4) Not applicable (not raised by parents, for example)	0

94) Did your parents separate or divorce before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

95) Did your father die before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

96) Did your mother die before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

97) Has there been a prolonged absence of your father before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

98) Has there been a prolonged absence of your mother before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

99) Have you frequently seen your father being drunk before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

100) Have you frequently seen your mother being drunk before you were twenty?

(1) Yes	1
(2) No	0
(3) Not applicable	0

101) Are you often bored? If so, when and where in general?

(1) No	0
(2) At work	2
(3) During my free evenings (home or elsewhere)	2
(4) During the week-ends (home or elsewhere)	1
(5) Not applicable	0

102) What do you usually do to relax?

(1) Nothing	2
(2) Sleep	2
(3) Go out, dance, go to parties	0
(4) Go visiting, receive visitors	0
(5) Sports	0
(6) Fishing	0
(7) Drink alcoholic beverages	2
(8) Play games (dominoes, card-games, etc.)	0
(9) Play music	0
(10) Watch television, listen to the radio, play records	0
(11) Not applicable	0

* 103) Are there any members of your family who, through their repetitive excessive drinking, cause damage to their own health or create problems in their home or at work?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 104) If so, who is the one fitting best into this picture?

(1) You	0
(2) Your (marital) partner	0
(3) One or more of your sons	2
(4) One or more of your daugthers	2
(5) Your father	0
(6) Your mother	0
(7) One or more of your brothers	2
(8) One or more of your sisters	2
(9) You certainly not	0
(10) Not applicable	0

105) And which one of the following relatives?

(1) Your father's father	2
(2) Your father's mother	2
(3) One or more of your father's brothers	2

(4) One or more of your father's sisters 2
(5) Your mother's father 2
(6) Your mother's mother 2
(7) One or more of your mother's brothers 2
(8) One or more of your mother's sisters 2
(9) Not applicable 0

106) Do you consider yourself someone who finds it extremely difficult to make contact with other people?
(1) Yes 1
(2) No 0
(3) Not applicable 0

107) Do you feel best in the company of others?
(1) Yes 0
(2) No 1
(3) Not applicable 0

* 108) Do you regularly use marihuana or hashish?
(1) Yes 1
(2) No 0
(3) Not applicable 0

* 109) Do you regularly use amphetamines ("speed", STP, etc.)?
(1) Yes 1
(2) No 0
(3) Not applicable 0

* 110) Do you regularly use cocaine (coca, "coke", etc.)?
(1) Yes 1
(2) No 0
(3) Not applicable 0

* 111) Do you regularly use LSD ("acid", etc.) or mescaline?
(1) Yes 1
(2) No 0
(3) Not applicable 0

* 112) Do you regularly use opium, morphine or pethidine?

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 113) Do you regularly use heroin?

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 114) Do you regularly use barbiturates (sleeping pills, soneryl, etc.)?

(1) Yes 1
(2) No 0
(3) Not applicable 0

* 115) What is your opinion of the activities of Alcoholics Anonymous (AA) on behalf of alcoholics?

(1) I am not acquainted with AA
(2) It doesn't interest me
(3) AA makes men unmanly
(4) AA has no success at all
(5) AA is only moderately successful
(6) AA is quite successful
(7) Not applicable

116) Does your wife resemble your mother in behavior or character?

(1) Yes 1
(2) No 0
(3) Not applicable 0

117) Does your husband resemble your father in behavior or character?

(1) Yes 1
(2) No 0
(3) Not applicable 0

118) Are you a legitimate child?

(1) Yes	0
(2) No; born out of wedlock	1
(3) Not applicable	0

119) Are or were your parents married?

(1) Yes	0
(2) No	1
(3) Not applicable	0

119a) Did your parents live in common-law?

(1) Yes	1
(2) No	0
(3) Not applicable	0

120) Are your parents relatives of one another?

(1) Yes	1
(2) No	0
(3) Not applicable	0

121) Have you been raised:

(1) For the greater part by your parents?	0
(2) Or for the greater part by fosterparents?	1
(3) Or partly by your parents and partly by fosterparents?	1
(4) Not applicable	0

122) Have you been raised by:

(1) Your mother	0
(2) Stepmother, fostermother	1
(3) Someone else	1
(4) Not applicable	0

123) Have you been raised by:

(1) Your father	0
(2) Stepfather, fosterfather	1
(3) Someone else	1
(4) Not applicable	0

124) How was the relationship between your parents or those who raised you?

(1) Indifferent, apathetic	1
(2) Harmonious, pleasant	0
(3) Calculating, cool, reserved	2
(4) Veiled conflicts	3
(5) Periodic outbursts of open conflict	4
(6) Frequent open conflicts	5
(7) Not applicable (grown up in an institution for example)	0

125) Are you regularly plagued by matrimonial conflicts?

(1) Yes	1
(2) No	0
(3) Not applicable	0

126) Are you regularly plagued by problems with your difficult children?

(1) Yes	1
(2) No	0
(3) Not applicable	0

127) Are you regularly plagued by money problems, debts?

(1) Yes	1
(2) No	0
(3) Not applicable	0

128) Are you regularly plagued by difficulties with your superiors at work?

(1) Yes	1
(2) No	0
(3) Not applicable	0

129) Are you regularly plagued by problems with your parents?

(1) Yes	1
(2) No	0
(3) Not applicable	0

130) Are you regularly plagued by problems with your in-laws?

(1) Yes	1
(2) No	0
(3) Not applicable	0

131) How is the relationship between you and your partner?

(1) Harmonious, pleasant	0
(2) Indifferent, apathetic	1
(3) Calculating, cool, reserved	2
(4) Veiled conflicts	3
(5) Periodic outbursts of open conflict	4
(6) Frequent open conflicts	5
(7) Not applicable	0

132) Are your parents:

(1) Both Arubans?	2
(2) Or is one of them Aruban?	1
(3) Or is neither of them Aruban?	0
(4) Not applicable	0

133) Have you been raised according to Aruban custom?

(1) Yes	2
(2) Partly	1
(3) No	0
(4) Not applicable	0

134) How was the relationship between you and your parents, c.q. those who raised you, during your childhood?

(1) Harmonious, pleasant	0
(2) Indifferent, apathetic	1
(3) Calculating, cool, reserved	2
(4) Veiled conflicts	3
(5) Periodic outbursts of open conflict	4
(6) Frequent open conflicts	5
(7) Not applicable	0

135) How is the relationship between you and your children?

(1) Harmonious, pleasant	0
(2) Indifferent, apathetic	1
(3) Calculating, cool, reserved	2
(4) Veiled conflicts	3
(5) Periodic outbursts of open conflict	4
(6) Frequent open conflicts	5
(7) Not applicable	0

* 136) Do you think that people in Aruba drink excessively, and if so, to what do you attribute this?

(1) People do not drink excessively in Aruba	0
(2) The high standard of living	0
(3) The hot climate	0
(4) Alcohol is cheap here	0
(5) Alcohol is a symbol of virility in Aruba	0
(6) Alcohol is a symbol of being adult in Aruba	0
(7) The Aruban custom of showing hospitality by offering drinks	1
(8) No opinion/don't know/not applicable	0

137) How was or is the attitude of your father towards your drinking habits?

(1) Disapproving	4
(2) Indifferent	3
(3) Ready to help	1
(4) Indulgent	2
(5) Not applicable	0

138) How was or is the attitude of your mother towards your drinking habits?

(1) Disapproving	4
(2) Indifferent	3
(3) Ready to help	1

(4) Indulgent 2
(5) Not applicable 0

139) How was or is the attitude of your (marital) partner towards your drinking habits?
(1) Disapproving 4
(2) Indifferent 3
(3) Ready to help 1
(4) Indulgent 2
(5) Not applicable 0

140) How was or is the attitude of your children towards your drinking habits?
(1) Disapproving 4
(2) Indifferent 3
(3) Ready to help 1
(4) Indulgent 2
(5) Not applicable 0

141) How was or is the attitude of your siblings towards your drinking habits?
(1) Disapproving 4
(2) Indifferent 3
(3) Ready to help 1
(4) Indulgent 2
(5) Not applicable 0

* 142) Which man is appreciated most by the Aruban woman, in your opinion?
(1) Aruban 0
(2) Bonairean 2
(3) Curaçaoan 2
(4) Windward Islands man 2
(5) Surinamer 2
(6) (European-) Dutch 2
(7) Venezuelan 2
(8) Latin-American 2
(9) American (U.S.A.) 2
(10) Not applicable 0

* 143) Which woman is most appreciated by the non-Aruban male, in your opinion?

(1) His own country's woman	0
(2) Aruban	2
(3) Bonairean	0
(4) Curaçaoan	0
(5) Windward Islands woman	0
(6) Surinam woman	0
(7) (European) Dutch	0
(8) Venezuelan	0
(9) Latin-American	0
(10) American (U.S.A.)	0
(11) Not applicable	0

144) Have you ever considered committing suicide?

(1) Yes	1
(2) No	0
(3) Not applicable	0

145) Have you ever attempted to commit suicide?

(1) Yes	1
(2) No	0
(3) Not applicable	0

146) If you have ever sought help for what you now recognize as alcoholism or the result of alcoholism, to which of the following authorities did you go at that time?

(1) General practitioner	1
(2) Neurologist	1
(3) Other medical specialist	1
(4) Priest, vicar, rabbi	1
(5) Social worker	1
(6) AA-member	1
(7) Probation officer	1
(8) Not applicable	0

147) Were you told at the time that you were an alcoholic?

(1) Yes	1
(2) No	0
(3) Not applicable	0

148) Were you told at the time that you were no alcoholic?

(1) Yes	0
(2) No	1
(3) Not applicable	0

149) Did they refuse you help or treatment at that time?

(1) Yes	0
(2) No	1
(3) Not applicable	0

150) Did you get from these contacts:

(1) Sufficient help	1
(2) Temporary help	1
(3) No help at all	1
(4) Deterioration of your condition	1
(5) Not applicable	0

151) Do you now consider yourself a member of AA?

(1) Yes	1
(2) No	0
(3) Not applicable	0

152) Do you speak at AA-meetings?

(1) Always	1
(2) Regularly	1
(3) Occasionally	1
(4) Never	0
(5) Not applicable	0

153) Do you ever go visiting or helping alcoholics in hospital, or alcoholics having trouble at home?

(1) Regularly	1
(2) Occasionally	1
(3) Never	0
(4) Not applicable	0

154) How often do you attend AA-meetings?

(1) More than 3 times a week	1
(2) 1-2 times a week	1
(3) 1-2 times a month	1
(4) Monthly	1
(5) 1-6 times a year	1
(6) Seldom	1
(7) Never	0
(8) Not applicable	0

* 155) How do you see an alcoholic?

1) As an emotionally disturbed person
(2) As a socially degraded person
(3) As a morally depraved person
(4) As an individual with a weak will
(5) As a patient
(6) Not applicable

156) How did you think of members of AA, before you joined AA?

(1) As religious fanatics
(2) As socially degraded individuals
(3) As sick, but otherwise healthy people
(4) As a secret society of drinking drunks
(5) Not applicable

157) What was your intention when you joined AA?

(1) To search inner peace
(2) To stop drinking
(3) To learn drinking "normally"
(4) Not applicable

* 158) Have you ever tried to stop drinking completely during a longer or shorter period, just to prove that you can keep away from alcohol?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 159) Have you ever quarrelled or fought with relatives or friends as a result of your drinking habits?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 160) Have you ever had problems at your work as a result of your drinking habits?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 161) Have you ever had money-problems as a result of your drinking habits?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 162) Have you ever been cast out by your family as a result of your drinking habits?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 163) Do you daily long for a strong drink, a beer or a glass of wine at the same time of the day?

(1) Yes	1
(2) No	0
(3) Not applicable	0

* 164) Does your drinking render your home life unhappy?

(1) Yes **1**
(2) No 0
(3) Not applicable 0

* 165) Do you prefer drinking alone?

(1) Yes 1
(2) No 0
(3) Not applicable 0

166) Have you, since your last hospitalization or treatment for alcoholism:

(1) Not taken one single drink 1
(2) Made a "slip" once 1
(3) "Slipped" several times 1
(4) Been drinking as much or more than before 1
(5) Not applicable 0

167) Have the difficulties at home and at work, since your last treatment for alcoholism:

(1) Improved 1
(2) Remained as serious 1
(3) Become worse 1
(4) Not applicable 0

* 168) Do you regularly experience an uncontrollable longing for a drink - even when you have not been drinking for quite a time - a desire you cannot, or only with an enormous effort, suppress? ("craving")

(1) Yes 1
(2) No 0
(3) Not applicable 0

169) Did you consider the example your father gave you in your childhood:

(1) A worthy goal, but impossible to realize for you
(2) A worthy goal, acceptable and attainable
(3) A bad example, objectionable

(4) Indulgent, too soft-hearted, overprotective
(5) Not applicable (father absent, for example)

170) Did you consider the example your mother gave you in your childhood:
(1) A worthy goal, but impossible to realize for you
(2) A worthy goal, acceptable and attainable
(3) A bad example, objectionable
(4) Indulgent, too soft-hearted, overprotective
(5) Not applicable (mother absent, for example)

* 171) Have you ever taken a drink just to please your friends or other people?

(1) Never	0
(2) Occasionally	1
(3) Regularly	1
(4) Always	1
(5) Not applicable	0

* 172) Have you ever taken a drink for fear of losing your friends or under pressure of your friends?

(1) Never	0
(2) Occasionally	2
(3) Regularly	2
(4) Always	2
(5) Not applicable	0

173) How often have you drunk *after* your last (hospital or outpatient clinic) treatment for alcoholism?

(1) Never	1
(2) Seldom (once a year or less)	1
(3) Only on festive days	1
(4) Once every two months	1
(5) At most once a week	1
(6) Every week-end	1
(7) 2-3 times a week	1
(8) Every day	1
(9) Not applicable	0

* *Questions asked in survey A and survey B & C.*

APPENDIX B

PARENT BEHAVIOR INVENTORY (P.B.I.) AND FOUR FACTOR-ANALYTICALLY CONFIRMED FACTORS

Responses and scores per variable.

Response	*Score*
1) That's exactly the way my father was	1
2) My father was more or less like that	2
3) My father wasn't really like that	3
4) My father was not like that at all	4

Variables	P B I -1	P B I -2	P B I -3	P B I.-4
1) My father always wanted to know exactly where I was and what I was doing.			0 4986	
2) My father decided which friends I was allowed to have.			0 5354	
3) My father forgot very quickly things he had forbidden me.				0.5519
4) My father didn't speak to me anymore when I had done something he didn't like.				
5) My father was more interested in my good than in my bad qualities.				
6) My father didn't allow me to go everywhere for fear of something happening to me			0 5892	
7) My father thought my ideas ridiculous.				0.4832
8) My father was very strict with me.	0.4367		0 6279	
9) My father always told me how I had to behave.			0.4317	
10) My father liked it when I brought friends home		0 4399		
11) My father always spoke to me in a kind voice.		0 5334		
12) My father was always thinking of things with which he could please me.		0 6220		
13) My father thought I was a real nuisance.				
14) My father told me often how much he loved me.		0 5969		
15) My father always asked what we had been doing at school and while playing outside.		0 5773		

16) One day I was punished for something I was allowed to do the next day				0 4020
17) I came off lightly when I had done something wrong				
18) My father understood my worries and problems		0 6444		
19) My father regretted my growing older and spending more time out of the house				
20) My father thought I was ungrateful when I didn't obey	0 5462			
21) My father was tolerant when I had misbehaved				
22) My father liked it when I decided by myself what to do and how to do it			—0 4223	
23) I don't believe he often thought of me				0 4325
24) My father sometimes wished he had no children				
25) According to him I would be punished later for everything I was doing wrong	0 5115			
26) My father kissed me often and he often caressed me		0 6457		
27) My father would not quickly forget things I had done wrong				
28) My father couldn't say no when I wanted something				
29) My father punished me harshly	0 5583			
30) My father gave me all the freedom I wanted			—0 4717	0 4800
31) My father saw to it that I came immediately home from school and that I was punctual for dinner			0 4460	
32) My father always listened to my opinions and ideas		0 6070		
33) He said how much I made him suffer	0 4038			
34) I was allowed to go anywhere without having to ask him first			—0 5156	
35) My father liked to do all sorts of things together with me		0 5146		
36) He regarded me as the most important thing in his life		0 6272		
37) My father would get angry about the least little thing I'd do wrong	0 4477			0 4196
38) My father wanted to know *where* I had been and with *whom* I had been out			0 6371	

39) My father ignored me completely when he was angry with me.				
40) My father raised hell when he was angry with me.		0.6343		
41) My father often praised me.		0.5817		
42) My father often said that I'd do as he told me if I loved him.				0.4150
43) He didn't oblige me to do things I didn't want to.		0.6016		
44) My father tried to understand me.	0.6376			
45) My father said that I would regret my behavior later on.	0.5923			
46) My father said I was getting on his nerves.	0.4062		0.4341	
47) My father wanted me to do exactly as he told me.	0.5556			
48) It depended on his mood whether something was allowed or not.				
49) My father hardly knew how I was doing at school.				
50) My father didn't like me to spend a lot of time out of the house.				
51) My father wanted to control everything I did.			0.6424	
52) I felt at ease in my father's presence.		0.5188		
53) My father kept at me until I did what he wanted me to do.	0.6158			
54) When I was small he cuddled me or gave me a kiss when I went to bed.		0.6745		
55) My father said that I'd sooner or later be punished for my behavior.	0.7407			
56) My father wished I were someone else.	0.6042			
57) My father seemed to be proud of what I was doing.		0.5999		
58) My father spent all his spare time with his children.		0.6361		
59) My father was very interested in what I was learning at school.		0.4464		
60) My parents allowed me the same things my contemporaries were allowed.			—0.4505	
61) It was easy to talk my father round.			—0.4634	
62) It seemed as if he was pleased when I was staying somewhere else for a while.			—0.4276	

63) When I upset him, he didn't want to have to do anything with me until I had made it up with him somehow.	0.6096			
64) My father loved me the way I was; he didn't try to change me.		0.5026		
65) He often gave me the feeling that nobody loved me.				0.5279
66) My father punished me so often that I no longer knew what I should or shouldn't do.	0.5192			
67) My father didn't let me make my own decisions.			0.4521	0.4122
68) My father let me have my way.			—0.6634	
69) He rather tried to be a friend than to boss me.				
70) My father always talked about what I was doing wrong.	0.5436			
71) My father was not interested in my friends.				
72) He allowed me to do everything I liked to do.			—0.5036	

APPENDIX C

METHODS TO EVALUATE THE PREDISPOSING FACTORS

Factor	Variable No.	Scores	Score for dichotomization in Risk Factor Analysis
1) *Paternal inebriety in one's youth*	99	1	>0
2) *Maternal inebriety in one's youth*	100	1	>0
3) *Interparental conflicts in one's youth*	124	1, 2, 3, 4, 5	>0
4) *Parent-child conflicts in one's youth*	134	1, 2, 3, 4, 5	>0
5) *Parental absence/broken home*			
5) a. Education in foster home	121	1	score 1 for 1 of the 3 variables
5) b. Education by foster mother	122	1	
5) c. Education by foster father	123	1	
6) *Absence of father in one's youth*	97	1	>0
7) *Absence of mother in one's youth*	98	1	>0
8) *P.B.I.-1; paternal punitiveness*		<median	<median
9) *P.B.I.-2; paternal affection/vs. rejection*		>median	>median
10) *P.B.I.-3; paternal control-dominance*		<median	<median
11) *P.B.I.-4; paternal rejection-indifference*		<median	<median
12) *Low social status of father*	K	6, 7	>5
13) *Death of father in one's youth*	95	1	>0
14) *Death of mother in one's youth*	96	1	>0
15) *Parental divorce in one's youth*	94	1	>0
16) *Illegitimacy*	119a	1	>0
17) *Parental consanguinity*	120	1	>0
18) *Familial inebriety*	104	2	>1
19) *Inebriety in extended family*	105	2	>1
20) *Low educational level*	20	5, 6	>4
21) *Rural domicile*	1	2	>1
22) *Autochthony*			
22) 1. Nationality	4	1	score ⩾1 for each of the 3 variables
22) 2. Aruban parents	132	1, 2	
22) 3. Aruban education	133	1, 2	
22a) *„Genetic" autochthony*	132	2	>1
23) *Male sex*	5	2	>1
24) *Economic availability versus poverty*			
24) a. Actual work situation	10	2, 3	<2
24) b. Subjective view of one's income	11	4, 5	or: <4
24) c. Income per month, in NAfl.	12	4, 5	or: <4

APPENDIX D

METHODS TO EVALUATE THE PERPETUATING FACTORS

Factor	Variable No.	Scores	Score for dichotomization in Risk Factor Analysis
25) *Marital conflicts*			
25) a. Regular marital conflicts	125	1	score ⩾1 for 1 of both variables
25) b. Conflicts with marital partner	131	1, 2, 3, 4, 5	
26) *Conflicts with one's children*	135	1, 2, 3, 4, 5	>0
27) *Low social status of respondent*	L	6, 7	>5
28) *Poor housing conditions*	8	2, 3	>1
29) (*Unmarried*) *civil state*	6	2, 3	>1
30) *Violation of one's confidence*			
30) a. By one's fiancée/marital partner	85	1	score 1 for 1 of the 6 variables
30) b. By one's father	86	1	
30) c. By one's mother	87	1	
30) d. By one's children	88	1	
30) e. By one's relatives	89	1	
30) f. By one's friends	90	1	
31a) *Authority conflicts with superiors at work*	128	1	>0
31b) *Authority conflicts with one's parents*	129	1	>0
31c) *Authority conflicts with one's parents-in-law*	130	1	>0
32) *Negative attitudes towards one's drinking*			
32) a. By one's father	137	2, 3, 4	score >1 for 1 of the 5 variables
32) b. By one's mother	138	2, 3, 4	
32) c. By one's marital partner	139	2, 3, 4	
32) d. By one's children	140	2, 3, 4	
32) e. By one's brothers and sisters	141	2, 3, 4	
33) *Social normativity*			
33) a. Relative's occasional inebriety	46	2, 3	<median (factor-analytical factor scores)
33) b. Relative's occasional being tipsy	47	2, 3	
33) c. Relative's being drunk weekly	48	2, 3	
33) d. Relative's drinking with meals	49	2, 3	
33) e. Relative's drinking without inebriety	50	2, 3	
33) f. Relative's being drunk every evening	51	2, 3	
33) g. Relative's abstinence	52	2, 3	
34) (*Proneness to*) *social pressure to drink*			
34) a. Drinking to please friends	171	1	score 1 or: score 2
34)b. Drinking for fear of losing friends	172	2	

35) *Hospitality drinking*	136	1	>0
36) *Stress at work*			
36) a. How many daily working hours	13	3	score >2 for 2 of the 3 variables
36) b. Do you enjoy your work	14	3	
36) c. A lot to do at work	15	3	
37) *Sociocultural deprivation autochthonous males*	142	2	>1
38) *Sociocultural deprivation heterochthonous females*	143	2	>1
39) *(Low) religious involvement*			
39) a. Kind of religion	7	1	score ⩾1 for 2 of the 3 variables
39) b. Frequent thinking about religion	79	1	
39) c. Frequent church attendance	80	1, 2, 3, 4	
40) *Anxiety*	77	1	>0
41) *Suicidality*			
41) a. Considered committing suicide	144	1	score 1 for 1 of both variables
41) b. Attempted suicide	145	1	
42) *Boredom*	101	1, 2	>0
43) *Introversion*	106	1	>0
44) *Dissatisfaction*			
44) a. Dissatisfaction with one's achievements	16	2, 3	score >1 for 2 of the 3 variables
44) b. Dissatisfaction with one's health	17	2, 3	
44) c. Dissatisfaction with one's living space	18	2, 3	
45) *Oral fixation or regression*			
45) a. How many cups of coffee/tea daily	21	2, 3	>median (factor-analytical factor scores)
45) b. How often consumption of sweets	22	2, 3, 4	
45) c. How often proneness to sweets	23	2, 3, 4	
45) d. What do you smoke	24	1	
45) e. How much do you smoke daily	25	1, 2, 3, 4	
46) *Drugs*			
46) a. Marihuana	108	1	score >0 for 1 of the 7 variables
46) b. Amphetamines	109	1	
46) c. Cocaine	110	1	
46) d. LSD	111	1	
46) e. Morphine	112	1	
46) f. Heroin	113	1	
46) g. Barbiturates	114	1	

APPENDIX E

CLASSIFICATION OF THE OBSERVED PROFESSIONS IN SURVEY B & C ACCORDING TO THE G.T.E., ADAPTED TO ARUBAN CIRCUMSTANCES

	Score
Social Class 1 (G.T.E. 07)	1
Teacher of Spanish language at high school	
Social Class 2 (G.T.E. 06)	2
Business man with employees	
Social Class 3 (G.T.E. 05)	3
Teacher	
Business man without employees	
Retail dealer	
Social Class 4 (G.T.E. 04)	4
Cashier	
Laboratory analyst	
Assistant laboratory analyst	
Chemical analyst	
Sergeant	
Police officer	
Pilot (in Oranjestad harbor)	
First mate	
Salesman	
Administrator	
Government official	
Clerk	
Lady secretary	
Jailer	
Station master at airport	
Custom house officer	
Fireman	
Personnel manager	

Social Class 5 (G.T.E. 03) 5

Welder
Carpenter
House painter
Pipe fitter
Mechanic
Motor mechanic
Baker
Barber
Shoemaker
Cook
Electrician
Butcher
Gas-fitter
Watch maker
Instrument maker
Fitter
Smith
Boiler maker
Furniture clother
Tailor

Social Class 6 (G.T.E. 02) 6

Laborer in the building trade
Executor at storehouse of public works department
Executor at airport department
Foreman at public works department
Foreman at cleansing department
Assistant pipe fitter
Engine driver
Postman
Bricklayer
Iron twister
Bill collector
Controller
Bar tender
Waiter

Storekeeper
Shop assistant
Crane operator
Telephone-operator
Assistant operator
Chauffeur
Employee at car rental enterprise
Captain of fishing boat
Night watchman
Baker's help
Fisherman
Sailor
Gardener
Fruiterer
Market merchant
Farmer

Social Class 7 (G.T.E. 01) 7
Unskilled laborer
Laborer
Dock laborer
Factory laborer
Laborer at public works department
Housemaid
Charwoman
Cleaner
Packer at printing office
Car washer
Laundry employee
Warehouseman at public works department
Prostitute
Refinery laborer
Aloe cutter

CURRICULUM VITAE

Oswald Raymond Wever, born October 7, 1939 in Aruba.

Elementary school at St. Dominicus College in Oranjestad. Gymnasium-β (secondary grammar school) at St. Petrus Canisius College in Nijmegen, from 1953 to 1958.

Medical school at the Catholic University in Nijmegen, graduated in 1968. General practice at Paradera, Aruba, from January 1969 to December 1972. Charged with the clinical and policlinical care for alcoholic, neurological and psychiatric patients in Aruba from October 1969 to September 1972.

Specialization in internal medicine December 1972 - December 1977: first year in Curaçao under supervision of Dr. L. W. Statius van Eps and Dr. A. Saleh, remaining years at the Department of Internal Medicine of the State University in Groningen headed by Prof. Dr. E. Mandema.

Previous publications on alcoholism mentioned in the bibliography.

STELLINGEN

I

Door toetsing van diagnostische criteria en (predisponerende/ perpetuerende) risicofactoren is het mogelijk patiënten in verschillende stadia van "pathologisch drinken" te herkennen, en tevens vroegtijdig "high risk" individuen en gezinnen - met betrekking tot het ontstaan van alcoholisme - op te sporen.

II

Phenotypisch alcoholisme op Aruba lijkt in belangrijke mate door exogene en minder door genetische factoren te worden bepaald, en moet daardoor goed toegankelijk kunnen zijn voor therapeutische maatregelen van buitenaf.

III

Klinische en poliklinische behandeling van alcoholisten en andere "pathologische drinkers" door een huisarts kan onder bepaalde omstandigheden even effectief zijn als specialistische behandeling.

IV

Het vervangen van een mislukte arterioveneuze fistel bij dialysepatiënten door een bovine graft is wegens de - op langere termijn - hoge mislukkingspercentages niet aanbevelingswaardig.

V

Endoscopische papillotomie als behandeling van choledocholithiasis is een goed alternatief voor chirurgische therapie, vooral bij patiënten met verhoogd operatierisico.

VI

Reversibele hypercortisolaemie met of zonder pseudo-Cushing syndroom kan optreden bij alcoholisme met of zonder chronische leverbeschadiging, hetgeen differentiaal-diagnostische consequenties heeft voor het Cushing syndroom.

VII

Bij insuline-afhankelijke diabetici dient men bij het toedienen van glucose-infusen en kaliumsparende diuretica rekening te houden met het optreden van hyperkaliaemie - ook bij afwezigheid van nierfunctiestoornissen, acidose of kaliumsuppletie.

VIII

De gelijktijdige bepaling van de serum-osmolaliteit met de vriespuntsverlagingsmethode èn met de gebruikelijke schattingsmethoden is - mits men braken, dehydratie, diabetes mellitus en nierinsufficientie uitsluit - een betrouwbare en snelle methode om de bloed-alcoholconcentratie te schatten.

IX

Aandoeningen van huid en slijmvliezen omvatten ongeveer 12 procent van alle aan de huisarts aangeboden ziekten en gezondheidsstoornissen, terwijl ca. 10 procent van de contacten tussen huisarts en patienten afwijkingen van de huid betreft. Op grond van deze gegevens kan de plaats van de dermatologie in de medische opleiding niet met de aanduiding „klein vak" worden omschreven.

X

Een gemiddelde dagelijkse consumptie van drie of meer standaardglazen bier, wijn of gedistilleerd, is een risicofactor voor hypertensie, hetgeen consequenties dient te hebben voor de aan hypertonici en excessive drinkers te geven medische adviezen.

XI

Alcoholisme bij aanstaande moeders kan aanleiding geven tot het ontstaan van groeiachterstand, cardiovasculaire afwijkingen, mentale retardatie, craniofaciale anomalieën en afwijkingen aan de ledematen bij de foetus: het foetale alcohol syndroom.

XII

Bij de selectieve distale splenorenale shuntoperatie volgens Warren ter preventie van recidiverende bloedingen uit oesophagusvarices, is het gewenste effect op de portale stroomrichting matig.

XIII

Bij alcoholische levercirrhose zijn nieraandoeningen beschreven, karakteristiek voor IgA-nephritis.

XIV

Verschillen in taken en functies tussen huisarts en specialist betreffen zowel de inhoud als de methoden van beider vakgebieden. Enerzijds verschilt het morbiditeitspatroon in de huisartspraktijk aanzienlijk van dat in het ziekenhuis, anderzijds is de benadering door de huisarts, in tegenstelling tot die van de klinisch specialist, meer gericht op problemen en minder op ziekten.

XV

Een bevolkingsonderzoek betreffende de prevalenties van bloedgroepen en andere "genetic markers" op Aruba heeft niet alleen medisch-wetenschappelijke betekenis, maar vormt ook een onmisbare bijdrage aan het ontdekken van de eigen identiteit.

XVI

Er zijn aanwijzingen, dat het medische beroep een risicofactor voor alcoholisme kan zijn.

Stellingen

behorende bij het proefschrift van

O. R. WEVER

Alcoholism in Aruba

Nijmegen 1977